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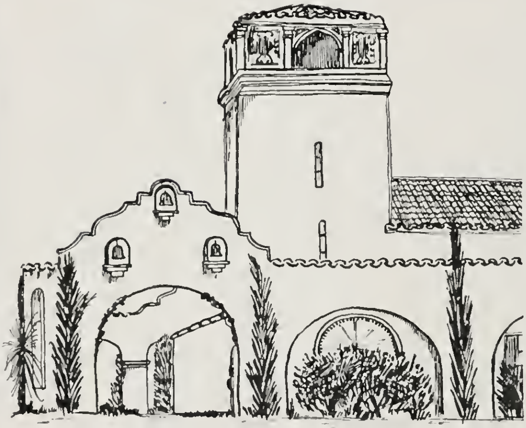

COLLEGE OF OSTEOPATHIC PHYSICIANS
AND SURGEONS • LOS ANGELES, CALIFORNIA

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Nothnagel's Practice

DISEASES
OF THE
BRONCHI, LUNGS
AND
PLEURA

BY

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AUTHORIZED TRANSLATION FROM THE GERMAN, UNDER THE
EDITORIAL SUPERVISION OF
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PREFACE.

THE excellence of the series of monographs issued under the editorship of Professor Nothnagel has been recognized by all who are sufficiently familiar with German to read these works, and the series has found a not inconsiderable proportion of its distribution in this and other English-speaking countries. I have so often heard regret expressed by those whose lack of familiarity with German kept these works beyond their reach, that I was glad of the opportunity to assist in the bringing out of an English edition. It was especially gratifying to find that the prominent specialists who were invited to co-operate by editing separate volumes were as interested as myself in the matter of publication of an English edition. These editors have been requested to make such additions to the original articles as seem necessary to them to bring the articles fully up to date and at the same time to adapt them thoroughly to the American or English reader. The names of the editors alone suffice to assure the profession that in the additions there will be preserved the same high standard of excellence that has been so conspicuous a feature in the original German articles.

In all cases the German author has been consulted with regard to the publication of this edition of his work, and has given specific consent. In one case only it was unfortunately necessary to substitute for the translation of the German article an entirely new one by an American author, on account of a previous arrangement of the German author to issue a translation of his article separately from this series. With this exception the Nothnagel series will be presented intact.

ALFRED STENGEL.

EDITOR'S PREFACE.

It has been a most congenial and profitable task that the distinguished editor of the American edition of Nothnagel's renowned System of Medicine put upon the writer. Fortunately for him, the eminent authors of the valuable monographs which make up this volume have by their breadth of learning, their exhaustive research, and, above all, by their extensive practical experience, made their essays so complete that he found but little to add. These additions include, among other things, some new matter on the anatomy and physiology of the bronchi; on foreign bodies in these tubes; on the pathology and bacteriology of bronchitis, and on the treatment of this affection. The reader will also find an account of the more recent researches on fibrinous bronchitis, or bronchiectasis, and on eosinophilia in asthma, as well as of Fraenkel's researches in the latter affection.

In the part on the Inflammations of the Lungs the writer upholds the view that pneumonia is the expression of a general pneumococcus infection, that the local pulmonary lesion is not a necessary part of the infectious process, and that the danger from this lesion is not as great as the danger from the toxemia. The recent work by Hutchinson and other authorities on the blood and on the urine in pneumonia has been incorporated, we trust with profit. The complications, prognosis, and treatment of this infection have been reconsidered, and the bacteriology of catarrhal pneumonia reviewed. The surgical treatment of abscess, gangrene, and other pulmonary conditions is brought up to date, and the author's views are emphatically indorsed. The employment of modern means of research, especially the use of the Röntgen rays for purposes of diagnosis, has been referred to and their sphere of usefulness defined.

In the section on Pleurisy will be found an account of the more recent bacteriologic studies; and notwithstanding the eminent author's forceful plea for the state of the soil—the tissues—as the essential factor in the etiology, we believe that the invading microorganisms must also be reckoned with in the pathology, diagnosis, and treatment of this affection. Here, too, will be found references to the recent studies of Morse on the leucocytes in pleurisy, to those of Williams and others on X-ray diagnosis, to the Litten phenomenon, and to other minor clinical facts.

With these additions it is hoped that the present volume will prove as valuable to us as to our colleagues on the Continent, and that thereby German Medicine will again receive the encomiums so willingly extended to it in the past by the English reading profession.

John Herr Musser.

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THE BRONCHI.

BY

FRIEDRICH A. HOFFMANN, M.D.

THE BRONCHI.

ANATOMY AND PHYSIOLOGY OF THE BRONCHI.

At the level of the fifth thoracic vertebra, as seen from behind, and of the second * intercostal space, as seen from in front, the trachea divides into its two main bronchi. The right bronchus is 2.4 cm. long, has a diameter of 2.3 cm., and shows six to eight plates of cartilage; the left is 5.1 cm. long, has a diameter of 2 cm., and shows nine to twelve plates of cartilage.† Both bronchi run sharply outward and downward, each into its own lung. The right runs under the arch formed by the vena azygos as it turns forward from the posterior chest-wall to the vena cava superior; the left, under the arch of the aorta. It may now be assumed, from the results of the investigations of Aeby, that each bronchus represents a trunk, which, becoming gradually thinner, gives off numerous branches, and may be followed as the main trunk to the finest divisions of the lowest and most posterior parts of each of the apices of the lungs. So each lung has a main bronchus which ramifies through it from the hilus to the neighborhood of the diaphragm. Practical anatomists understand under the name main bronchus, however, only that part which extends from the bifurcation of the trachea to the point where the first lateral branches are given off. In reality the main bronchus in its narrower portions cannot, without special methods, be distinguished from its branches; we therefore follow the old classification, and say that the main bronchus has a direct continuation, from which the lateral twigs are given off. We name this main bronchus, with its continuation, the bronchial trunk.

The right bronchus has a more nearly vertical course than the left, and a horizontal section through the lung therefore presents it more in cross-section, while the left is cut more longitudinally. The right bronchus extends forward to the right pulmonary artery; it is separated from the esophagus and from the right vagus by one or several lymph-glands. The left bronchus extends forward close to the left auricle, and the left pulmonary artery runs obliquely outward in front of it. Behind it lies the aorta descendens; above, the arch of the aorta; between it and the aorta descendens runs the

---

* As shown in Braune's Atlas.

† Reckoned according to Henle, "Eingeweidelehre."

left vagus, while to the outer side lie lymph-glands and large branches of the pulmonary artery. It is not quite contiguous to the esophagus, but is separated from it by small lymph-glands. At its origin, however, it lies adjacent to the esophagus.

The first branches of the bronchial trunk are given off at right angles; indeed, all the bronchi are branches and sub-branches of the two bronchial trunks.

Aeby called attention to a very important point when he studied the relations of the bronchi to the pulmonary arteries in man and many animals. The pulmonary arteries are found on the posterior aspect of the bronchi; hence each principal arterial branch, in order to reach the bronchi, must cross the bronchial tree. On the left side the artery crosses the main bronchus; on the right side, the artery crosses the bronchial trunk beyond the point where the first branch is given off. This relation, which is illustrated in the accompanying diagram (Fig. 1), Aeby declares to be a very important one; the lateral branch given off above the crossing is called eparterial, while all the others, which are given off beyond the crossing, are known as hyparterial branches. In the normal human subject there is one eparterial bronchus, which is on the right side of the body; all other bronchi are hyparterial. The hyparterial branches are given off in two rows along the main stem, one dorsal and one ventral. But these two rows are not exactly opposite each other, the ventral being somewhat more external and posterior, and thus crowding the dorsal.

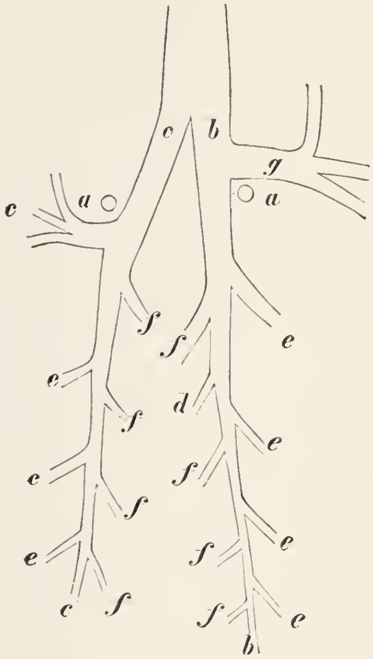

Fig. 1.—*a*, *a*, Arteriæ pulmonales; *b*, *c*, right and left bronchial trunk; *d*, cardiac bronchus; *e*, *e*, *e*, ventral hyparterial side-bronchi; *f*, *f*, *f*, dorsal hyparterial side-bronchi; *g*, eparterial bronchus.

The greater portion of the periphery of the bronchial trunk is free from branches, internally and anteriorly, and, in general, the dorsal bronchi are shorter and smaller than the ventral. This explains the fact that the bronchial trunk has far more lung tissue lying on its external and ventral, than on its internal and dorsal, sides.

The lateral bronchi have mostly a descending course, becoming more and more vertical. The eparterial branch is given off about midway between the bifurcation and the origin of the first hyparterial branch, but its position is very variable, and it may even arise from the trachea; in this way Aeby explains the rare cases of trifurcation of the trachea. The hyparterial bronchi are quite symmetric, and present on each side four ventral and four dorsal branches. On the right, however, a remarkable bronchus is very constantly found between the first and second dorsal branches; it is called the *heart bronchus* because its analogue in animals leads to a special lobe of the lung, the heart lobe. In man it branches in the inner and anterior portion of the lower lobe of the right lung, which lies next to the heart. The manner in which the bronchi divide has caused an anterior and a posterior (also ventral and dorsal) system to be distinguished, with ascending and descending branches in each. The former correspond to the portion of the lung the motion of which is relatively slight and which extends backward and downward; the latter to the movable anterior and peripheral portions of the lungs.

The cross-section of the trachea* is smallest just under the larynx and increases steadily down to about the center of the tube, where it reaches its maximum size. Then it decreases in size until it reaches a point 3 cm. above the bifurcation; it then increases again up to the point where it divides into the bronchi. This peculiar variation is repeated in the bronchi, so that the air-current undoubtedly enters with a marked rotatory motion. The cross-section of the right bronchus is larger than that of the left (the proportion is about 100 : 77.5). The sum of the cross-sections of the two bronchi at the point of bifurcation is greater than the sum of the cross-sections of all their branches. The left bronchus is diminished up to the point where the branches are given off. In general the size of the cross-section of a bronchus depends upon the size of the column of air which passes through it. Lung emphysema causes an increase in size, while pleural adhesions cause a narrowing of the lumen.

Further details we owe to Hasse.†

[The conclusions arrived at by Aeby in regard to the asymmetry of the lungs, which had won general acceptance and found their way into most of the current text-books on anatomy, have been called in question by Huntingdon.‡ He suggests that the left upper lobe is the equivalent of the right upper and middle lobes, the eparterial bronchus on the right side being the morphologic equivalent of the upper segment of the first secondary bronchus given off on the left side; the next ventral branch on the right side being the equivalent of the lower segment of the first secondary bronchus on the left side. Otherwise, the divisions of the bronchi are the same on both sides, with the exception of the cardiac bronchus, which is always found on the right side.

Aeby's hypothesis of the morphologic equivalence of the middle right and upper left lobe of the human lung is therefore incorrect. The proposition should read:

---

* Braune and Stahel, *Archiv für Anatomie und Physiologie*, 1886.

† *Archiv für Anatomie und Physiologie*, anatom. Abtheilung, 1892.

‡ "The Eparterial Bronchial System of the Mammalia," Geo. S. Huntingdon, *Annals N. Y. Acad. Sci.*, XI, No. 8, 1898.

Right Side.		Left Side.
Upper + middle lobe	=	upper lobe.
Lower + cardiac lobe	=	lower lobe.

Except for purposes of topography, the distinction of eparterial and hyparterial bronchi should be abandoned, at least to the extent of clearly recognizing the fact that in a symmetric lung every right "eparterial" bronchus finds its morphologic equivalent among the hyparterial bronchi of the left side. The divisions of the bronchial tree are described by Huntingdon as "practically dichotomous."

The **relations of the trachea and bronchi to the thoracic walls** have recently been worked out with the aid of the Röntgen rays by Blake.*

For convenience in description the following perpendicular lines are taken:

1. The median line.
2. The sternal line, along the border of the sternum.
3. The mammillary line, through the nipple.
4. The parasternal line, midway between the sternal and the mammillary line.

The trachea, which is in the median line at the lower part of the neck, lies in the right sternal line at its bifurcation, which corresponds to the right side of the center of the vertebræ posteriorly. The deviation of the trachea is caused by the aorta, which crowds it to the right side. It bifurcates at about the level of the intervertebral disc between the fourth and fifth thoracic vertebræ, which corresponds nearly in level to the tip of the fourth thoracic spine. This point is not absolute, as it is influenced by the respiratory movements and by the position of the neck and head. In the adult it is below the level of the scapular spine; on the anterior wall it is just internal to the junction of the lower border of the second costal cartilage with the sternum. In the child, on account of the horizontal direction of the ribs, and the consequent greater height of the sternum relative to the vertebræ, the point of bifurcation is under the right border of the sternum at the level of the third costal cartilage.

The stem bronchi, beginning at the bifurcation, end or become so small as to be negligible opposite a point on the posterior thoracic wall at the eighth rib, situated, in the adult, on the left side three inches, and on the right side two inches, from the vertebral spine; in the child one and one-half inches and one inch, respectively. On the anterior thoracic wall these points are situated, on the left side, at the

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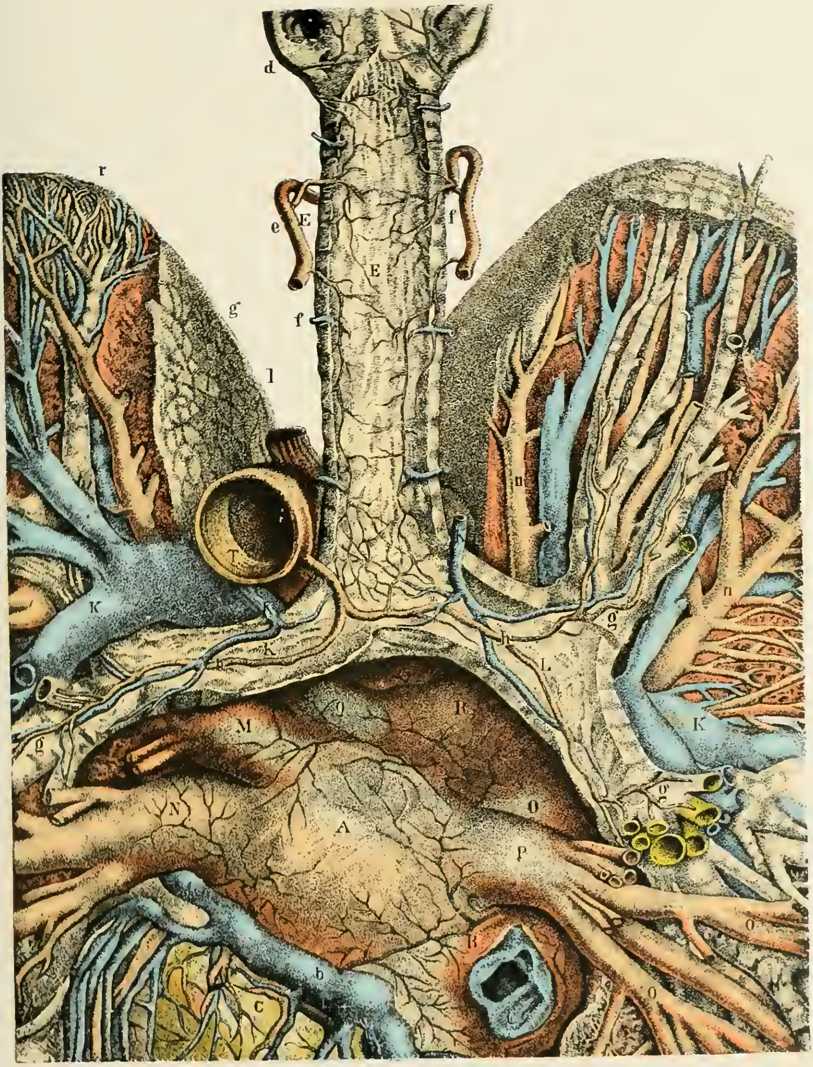
* "The Relation of the Trachea and Bronchi to the Thoracic Walls, as Determined by the Röntgen Rays," by Joseph A. Blake, M.D., *The American Journal of the Medical Sciences*, March, 1899.

PLATE I.



A. Left auricle.

B. Right auricle, with opening of inferior vena cava (C).

C. Left ventricle.

E. Trachea.

K. Left bronchus, pulmonary arteries in the lung, bronchial veins.

L. Right ventricle.

M, N, O, P. Pulmonary veins.

Q, R. Pulmonary arteries.

T. Aorta.

b. Coronary vein.

d. Superior thyroid vessels.

e, f. Inferior thyroid vessels.

g. Ramifications of the bronchi.

h. Superior bronchial arteries.

n. Pulmonary veins.

r. Apex of left lung.

fifth rib just internal to the mammillary line; on the right side, at the fifth rib in the parasternal line.

The course of the primary or stem bronchi in relation to the posterior and anterior walls is as follows:

On the posterior wall, in the adult, the course of the left bronchus is from a point to the right of the fourth thoracic spine to a point on the eighth rib, three inches to the left of the spine. The course of the right bronchus is from the same point above to a point on the eighth rib, two inches to the right of the spine.

On the anterior wall in the adult the course of the left bronchus is from the lower part of the second right chondrosternal articulation to a point on the fifth rib, just internal to the mammillary line; and of the right bronchus from the same point above to the intersection of the fifth rib with the parasternal line.

In children the point of commencement of the bronchi is opposite the third chondrosternal articulation; otherwise the course is the same as in the adult, due allowance being made in the distance of their termination from the vertebral spines posteriorly. In comparing the direction of the two bronchial trunks, the right is seen to correspond very closely with that of the trachea, while the left diverges markedly.

The relations of the first branches of the stem bronchi are approximately as follows, their position in regard to the chest-wall being influenced by the same factors which affect the position of the tracheal bifurcation:

The eparterial bronchus is given off at the level of the lower part of the second interspace or the third cartilage anteriorly, and at the level of the fifth space or sixth rib posteriorly. The first branch from the left bronchus is given off at the level of the third space or fourth cartilage anteriorly, and at the level of the sixth interspace or seventh rib posteriorly. These points may also be determined by measurements, the eparterial bronchus arising one inch, the first branch from the left bronchus two inches, from the tracheal bifurcation.

The points of origin of the first branches on the two sides differ greatly as to their distance from the median line, being anteriorly on the left side in the parasternal line, on the right midway between the sternal and parasternal lines; while posteriorly the point of origin of the eparterial bronchus is at the right border of the vertebral column, that of the first branch from the left bronchus being two and a half inches from the spines of the vertebræ.—Ed.]

The eparterial bronchus of the right side supplies the upper lobe of the right lung; the first ventral hyparterial bronchus is for the middle lobe; the other ventral as well as all the dorsal hyparterial branches go to the lower lobe. On the left side the first ventral hyparterial branch goes to the upper lobe.

Physiologically considered, the two lungs may be reduced to two cones, the apices of which both point inward, while the base of the upper turns upward and forward, that of the lower outward and backward.

From the lateral bronchi arise the secondary bronchi, which bear the same relation to the lateral bronchi that these do to the bronchial trunk.

[The successive divisions of the bronchial tree are therefore as follows: (1) Main trunk; (2) primary bronchi or first lateral branches; (3) secondary bronchi, given off from lateral branches; (4) tertiary bronchi, branches of secondary ("bronchus respiratorius" or "bronchial tube"); (5) terminal bronchus (ductulus alveolaris), the continuation of a tertiary bronchus.

W. S. Miller has recently made a careful study of the terminal relations of the bronchial tree to the pulmonary tissue, the results of which were published in the "Archiv für Anatomie und Physiologie," 1900. According to his description, the last division of the bronchus, before breaking up into the parenchyma of the lung, is known as the "*terminal bronchus*" (ductulus alveolaris). The distal extremity of each terminal bronchus widens out into an expansion in the walls of which are three to five circular openings which communicate by a short passage with a space or cavity—the "*atrium.*" A second passage leads from the latter into a second central cavity, the *air-sac*, or infundibulum, which is set about with small irregular cells, the *air-cells* or alveoli. From this description it will be seen that the air-sacs do not communicate directly with the terminal bronchus, as was formerly described, but between each air-sac and terminal bronchus there is a cavity, constant in all portions of the lung, for which Miller suggests the term "*atrium.*"

The terminal bronchus has an average diameter of 0.4 mm.; its walls contain abundant masses of smooth muscle-fibers and are lined with cylindric epithelium. A ring of unstriped fibers, resembling a sphincter, also surrounds the openings leading into the atria; but beyond this point no muscular tissue is found in the parenchyma of the lobule.

The atria have an average diameter of 0.28 mm., and are to be distinguished from the terminal bronchi by the absence of smooth muscle-fibers and the change which takes place in the epithelium; that of the terminal bronchi being cuboidal, while that of the atria is flat pavement epithelium. The diameter of the atrium is slightly more than half that of the air-sac or infundibulum, which is 0.41 mm.; and, unlike the latter, each atrium has three or more openings: one communicating with the terminal bronchus, the remaining ones with air-sacs. The air-sac, on the other hand, has only a single opening, that by which it communicates with the atrium. The walls of the atrium resemble those of the air-sac, being thin and devoid of muscle-fibers and having inclosed within them the capillary network of blood-vessels.

In addition to the air-cells (alveoli) surrounding the air-sacs, others are found arising from the atria, and yet others clustered about the smaller tertiary bronchi and the terminal bronchus. The latter are smaller and less numerous than those arising from the atrium and from the air-sac. No distinction in size has been found between the air-cells of the atrium and those of the air-sac.

Between the air-cells, on the central side of the air-sac, the ramifications of the pulmonary artery are found; on the peripheral side the ramifications of the pulmonary vein are seen, with its radicals running over the tips of the air-sacs.

That air-cells do not communicate Miller proved by interesting injection experiments on a lung, one lobule of which had been isolated by dividing its bronchial branch near the trunk. On injecting fluid into the main bronchi it was found that none would pass to that part of the lung supplied by the divided branch; the isolated portion of lung remained collapsed and sharply marked off from the distended portion.—Ed.]

In the region of the hilus of the lung the bronchial tree is fixed by bands of mediastinal tissue, designated by Teutleben *ligamenta suspensoria diaphragmatis*, which originate from the lower cervical and upper thoracic vertebræ on both sides as thickened bands of fascia prævertebralis, support the root of the lungs, and, in intimate relation with the pericardial sac, extend to the diaphragm.*

The bronchi, however, are nowhere fixed during respiration; even the bifurcation descends during inspiration, to rise again during expiration.

The bronchi become constantly narrower as more and more branches are given off; the tertiary branches still have a diameter of about 4 mm., but toward the end it becomes only 0.3 to 0.4 mm., and so they terminate in the infundibula, the approach into which from the bronchial lumen is somewhat constricted, and into which on the other side the alveoli open.

[Brosch † reports a case in which there were peculiar changes in the shape of the trachea and of the main bronchi. The patient was a man, sixty-three years of age, who suffered from right-sided facial paralysis, aphasia, and paralysis of the right upper extremity. He had the costal type of breathing. The laryngeal cartilage was partly ossified. The transverse section of the trachea presented a lyre-like appearance, and the ends of the tracheal cartilages were about 4 cm. apart. This change was present throughout the whole length of the trachea, and marked in the main bronchi, with less change in the larger bronchi, while the smaller bronchi showed a general enlargement. Longitudinally the main bronchi were flattened and the posterior membranous wall enormously stretched. The cross-section of the main bronchi was slit-like. Brosch compares the picture to that of a strung bow: the cartilage corresponding to the bow itself, and the membranous portion to the bow-string. The lumen of the bronchi was very small, as only the central portion of the cartilaginous rings was convex, while the outer portion of the rings was concave, so that the cartilage and membranous portion of the bronchial wall were united at a very acute angle. The circumference of the bronchi was 6.5–7.5 cm. (2.6–3 in.), while the lumen was narrowed, so that Brosch has called this condition *dilatatio paradoxa*. The trachea was lengthened as well as widened, and showed a slight tortuous curve. Brosch thinks that the change in the bronchi gave rise to the costal type of breathing, and thinks it probable that, together with the paralysis already present, there may have been some affection of the motor fibers of the recurrent nerve to account for the change in the structure of the trachea and bronchi.—Ed.]

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* *Archiv für Anatomie und Physiologie*, anatom. Abtheilung, 1877.

† *Deutsches Archiv für klinische Medicin*, vol. LXVIII, Nos. 3 and 4, p. 264.

The structure of the main bronchi is quite similar to that of the trachea; in front we find plates of cartilage, which insure that the lumen shall remain open, but this protection is lacking in the posterior portion. In regard to the finer histologic structure of the main bronchi, I would only have to repeat the description already given for the trachea.

As the diameter of the bronchi decreases, the walls naturally become thinner and more delicate. The epithelial cells become lower and lower, but cilia are still present up to near the terminal branches; only in the finest branches there is found a simple epithelium of cuboidal cells. Under this is the basement membrane with elastic fibers, which are partly circular and partly longitudinal; then follows the musculature, consisting in reality of a muscular ring of smooth muscle-fibers; finally, we find an adventitia of connective tissue containing a small amount of cartilage and well-developed elastic fibers. All these layers are found, from the largest bronchi down to the smallest, diminishing in size to correspond with the size of the bronchi.

The first change in the structure of the bronchial wall, as the size of the bronchi decreases, concerns the plates of cartilage, which, immediately after the division of the main bronchus, become irregular. The further we go, the smaller and more irregular they become and the further outward they are pushed, until they are present only as a smooth ring or half ring surrounding the mouth of a lateral twig or supporting the fibrous septum which separates the two branches of a bifurcation. In this form they are still to be found in bronchi of a diameter of 1 mm. In the places where the bronchi divide, the cartilage is found in the form of a perfect crescent, the concavity of which coincides accurately with the bifurcation, while the convexity is relatively thicker. This fact has been shown by King.*

The racemose glands are found singly as far down as is the cartilage (Henle).

The elastic tissue, studied by means of the orcein stain, is so arranged that a considerable amount is found just under the epithelium, which in thicker sections may appear like a dense membrane,

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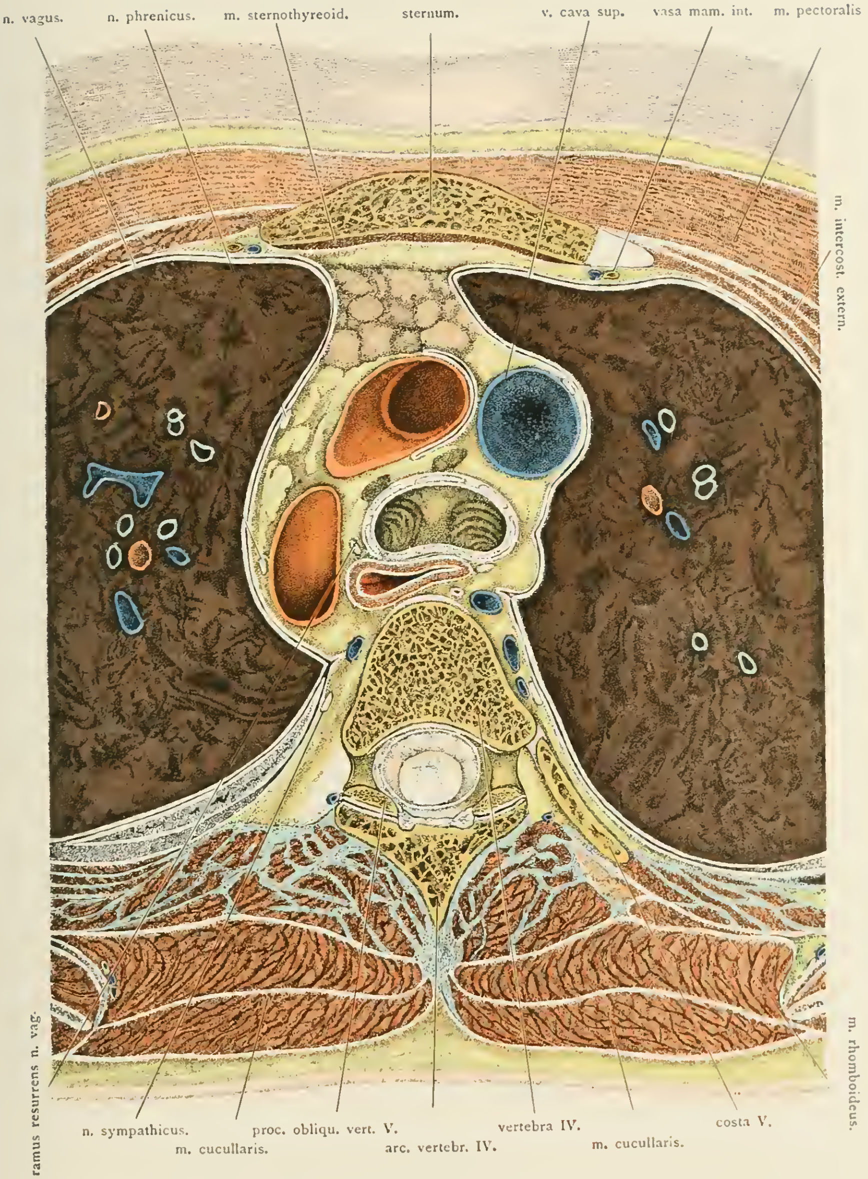
* *Guy's Hospital Reports*, Series I, vol. V, 1840.

PLATE 2.


n. vagus.
n. phrenicus.
m. sternothyreoid.
sternum.
v. cava sup.
vasa mam. int.
m. pectoralis ma
m. intercost. extern.
ramus resurrens n. vag.
n. sympathicus.
m. cucullaris.
proc. obliqu. vert. V.
arc. vertebr. IV.
vertebra IV.
m. cucullaris.
costa V.
m. rhomboideus.

but in reality is nothing but a network of coarse fibers, from which other fibers radiate into the underlying layer that contains the circular muscle-fibers. A robust layer of elastic fibers also surrounds each cartilaginous plate and the bronchus itself; two or three places are found in which the elastic tissue is collected into thick bundles, which accompany the bronchus along its entire length (Fig. 2, *c*, *c*, *c*). The smooth muscle-fibers are also accompanied by many elastic fibers, and in orcein preparations one gains the impression that the elastic fibers predominate. Besides the circular fibers and the rare bundles which, running more obliquely, ascend from the deeper cell layer to the circular fiber layer, some larger bundles are found coming from the smooth muscle-fibers and representing longitudinal bundles of fibers. There are very few glands to be found in this region of the bronchus, although there is still considerable cartilage, and on careful examination we find, in addition to the large plates of car-

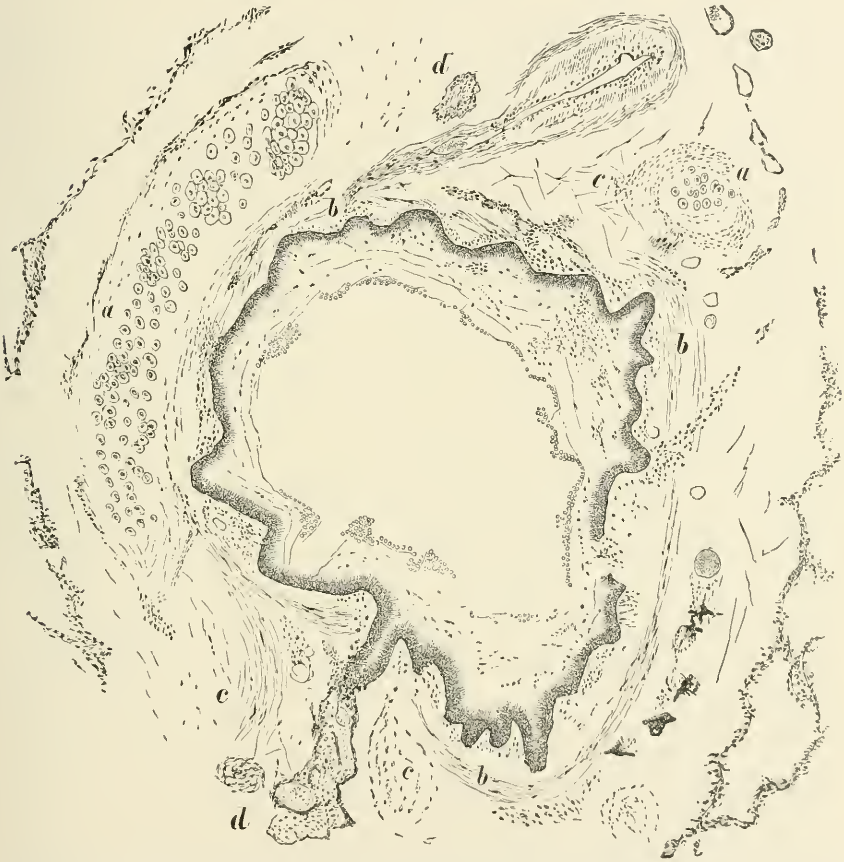


Fig. 2.—Cross-section of a normal bronchus of 1 mm. diameter: *a*, *a*, Cartilage; *b*, *b*, *b*, ring of smooth muscle tissue; *c*, *c*, *c*, longitudinal smooth muscle-fibers with much elastic tissue; *d*, *d*, glands.

tilage, some very small bits (Fig. 2, *a*, *a*); with the orcein stain, these are completely covered by the large elastic fibers, which run through and around them. The abundance of small blood-vessels just under the epithelium, as well as in the submucous tissue, is surprising, but it is no less surprising that only one large vessel, evidently an artery, is present; a corresponding vein is lacking. It is also surprising that the mucous membrane is by no means firmly attached to the cartilage, but is connected to it by loose connective tissue, through which ramify many elastic fibers and large vessels. A considerable development of lymph-vessels is also present, so that the mucous membrane is not firmly fixed to the cartilage, permitting the abundant layer of circular fibers which is found here to contract vigorously on the mucosa.

[Pearce * gives the following description of the distribution of elastic tissue in the normal bronchus, as determined by the use of Weigert's elastic stain: In the large bronchi a considerable amount is found in the submucosa and in the coarse connective tissue about the glands and cartilage; in the submucosa it is found in the loose connective tissue supporting the blood-vessels, appearing as a fine interlacing network which, near the basement membrane, shows a tendency to longitudinal arrangement. A few fibrils often penetrate between the acini of the mucous glands. About the cartilage coarser, dense, entangled fibers are found, the perichondrium staining so intensely as to appear composed almost entirely of elastica. As the bronchi become small, the amount rapidly diminishes until in the smallest bronchi it appears as fine, generally parallel, occasionally branching fibers. In the terminal bronchus the fibers are few in number, but increase at the vestibule, where they form deeply staining varicose swellings, sometimes appearing as small oval or round knobs of interlacing fibers.—Ed.]

I would also call attention to the mucus which covers the epithelial cells. It consists of round drops, which are distinctly recognizable. In places, large nucleated forms are found in them, which, after careful examination of the best preparations, I regard as degenerated and cast-off epithelial cells. On the surface of this mucus, blood-corpuscles even are found. (Polluted in the section.)

Arnold † describes in the wall of the normal bronchus of man, masses of round cells which he characterizes as lymphatic tissue. The knowledge of these masses is of importance to the investigator; they may easily be confused with pathologic infiltrations and with bundles of smooth muscle-fibers. It is important that Arnold found them in the peribronchial tissue, but not in the true bronchial wall. But the subepithelial connective tissue, in which, even in the healthy lung, the cells are found abundantly massed together, is in reality the same kind of tissue.

The smallest bronchi, called also bronchioles, are lined with cuboidal epithelium, under which is found a hyaline layer of distinct elastic tissue, and around this is a connective tissue rich in cells.

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* *Journal of Medical Research*, vol. I, No. 1, new series, 1901.

† *Virchow's Archiv*, Bd. LXXX, S. 315, 1880, Tafel IX.

These bronchioles pass directly over into the infundibula, into which the cuboidal epithelium also extends, but becomes gradually flatter. A smooth musculature is generally no longer present in the bronchioles. Authors find the non-striped muscle, however, up to the end of the bronchi, and Rindfleisch even mentions a thickened ring of it at the entrance into the alveolar duct. (See editor's note on p. 22.)

The great mass of the blood-vessels of the lung belongs, as is well known, to the pulmonary vessels, while those of the bronchi belong to the bronchial system. But the pulmonary artery accompanies the bronchus and in its course gives off numerous branches to the walls of the smaller bronchi. In like manner the pulmonary veins are formed in part by vessels which receive blood from the capillaries of the bronchial artery.* Rindfleisch has called especial attention to the peculiar terminal course of the pulmonary artery.†

These minute arterial twigs enter the acinus not far from the bronchus and just where the three main alveolar ducts are united in their origin, break up into a large number of intermediate branches which penetrate to about the middle of the acinus, and then resolve themselves into capillaries. They are thus intraacinous, while the veins are interacinous, so that the blood from the middle and root of the acinus passes outward.

The most detailed description of these relations is given, however, by Küttner.‡ The branches of the pulmonary artery, from the hilus of the lung to the lobule, are in constant apposition with the bronchus and about correspond to it in size. Throughout their course they give off large branches which penetrate to the limiting membrane and, in common with the relatively few branches of the bronchial artery, form a dense capillary network in the subepithelial layer. In the lobules of the lungs themselves it is peculiar how branches that are practically capillaries are given off from relatively large arteries. "In each pulmonary artery, one recognizes (in corrosion preparations), even with the unaided eye, the extremely fine arterial radicles as closely crowded fine hairs and bristles, which give to the surface of the main trunk a peculiar furry character. Some of these finest branches pass to the mucosa of the smallest bronchi or to the peribronchial connective tissue, but the greater part lose themselves in the capillary network of the alveoli. It follows, therefore, that the capillary region of the pulmonary artery is by no means strictly separated from that of the bronchial artery. The pulmonary veins arise from the capillaries of the alveoli and infundibula in the perilobular tissue and are interlobular in their course, separated from the artery and the bronchus. The pulmonary veins receive also numerous branches from the bronchial walls."

The bronchial arteries are characterized by a peculiarly winding course; at the end of the terminal bronchi the bronchial arteries also end, and their capillary region passes over into that of the pulmonary artery. No doubt blood from the bronchial artery reaches the infundibula and the alveoli.

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* Reisseisen, cited by Luschka.

† Ziemssen, "Handbuch," Bd. v, 2. Theil, S. 174.

‡ *Virchow's Archiv*, Bd. LXXIII, S. 487.

Each larger branch of the bronchi is accompanied by two or three branches of the bronchial artery, which communicate with each other.

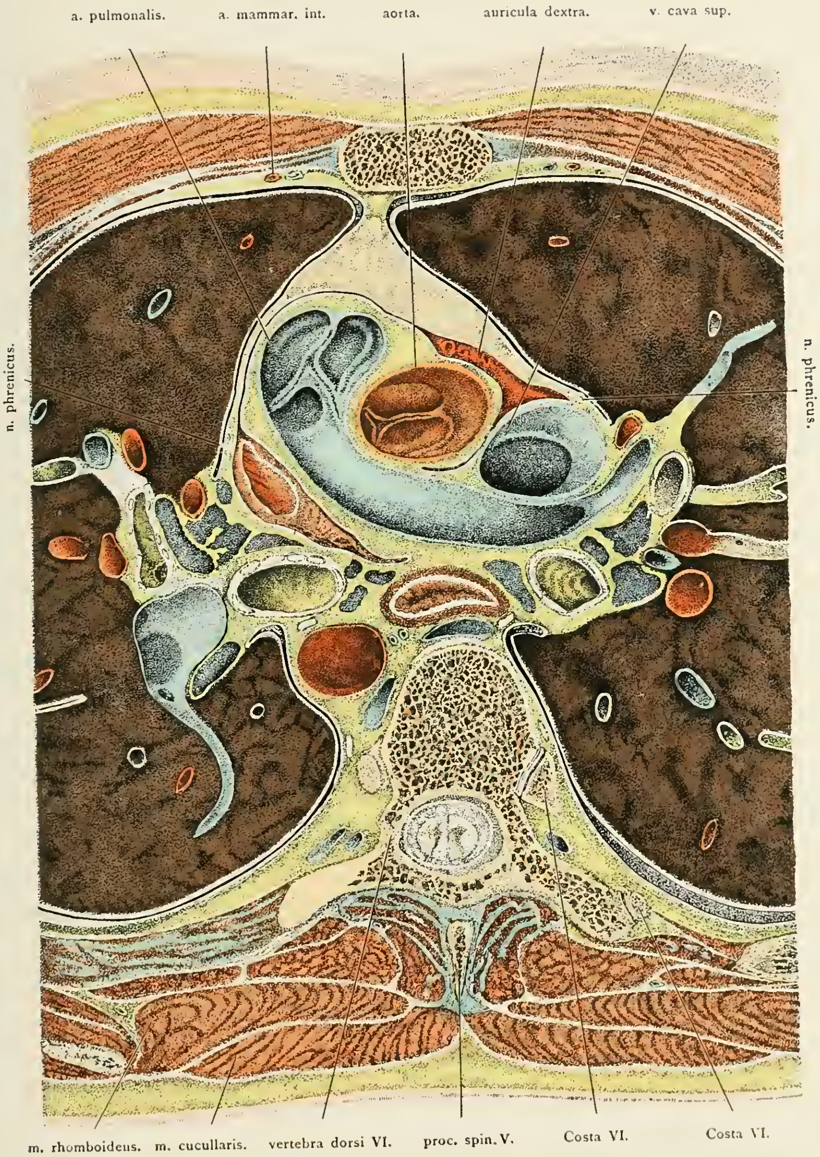
The bronchial veins receive their blood from the external layers of the bronchial wall as well as from the subpleural and interlobular capillary regions (from this latter region the pulmonary veins also are supplied).

The arteries which supply the lower end of the trachea, the two main bronchi, and the lymph-glands near the point of division, are the *superior bronchial arteries.* They arise either as a common branch, or as two branches from the lower wall of the arch of the aorta, where it winds over the left bronchus. The common branch divides into a right and a left bronchial artery, which then branch further with the bronchi. It also occurs, however, that branches arise from other arteries and in part take the place of those named: from the inferior thyroid artery, from the superior intercostal artery, from the internal mammary artery, from the internal mediastinal artery, and from the subclavian.

The numerous bronchial twigs are also supplied by branches arising from the aorta descendens at the level of the fourth and sixth dorsal vertebræ. The first is the common bronchial artery, which divides into a right and a left bronchial artery, and these enter the right and left lungs. The second is the second left bronchial artery, which goes only to the left lung. These branches wind about the bronchi and lose themselves in two capillary plexuses, one for the muscular coat and one for the mucosa; the latter, as we already know, probably receives a still greater contribution from the pulmonary artery. From these bronchial networks, especially those belonging to the smallest bronchi, numerous branches pass into the pulmonary veins.

The **bronchial veins** receive their blood from the external layers of the bronchial wall, from the subpleural and interlobular capillary regions, from the larger bronchi, and from the hilus of the lung. Finally they form two or three trunks, which, on the right, empty into the azygos or vena cava superior; and, on the left, into the superior intercostal vein or the left innominate vein. The capillaries of the bronchi have also two outlets: one into the right, and one into the left heart; the great majority of those from the smallest bronchi empty into the left heart, while those from the large bronchial trunks empty into the right heart. This fact may be of importance in understanding the hyperemia of the bronchi in affections of the heart. The bronchial veins are distinguished from all the other veins of the body by the fact that the circulation in them is not supported by the suction power of inspiration.

PLATE 3.


a. pulmonalis.
a. mammar. int.
aorta.
auricula dextra.
v. cava sup.
n. phrenicus.
n. phrenicus.
m. rhomboideus.
m. cucullaris.
vertebra dorsi VI.
proc. spin. V.
Costa VI.
Costa VI.

[The pulmonary artery follows the bronchus closely throughout its entire length, and when it reaches the last forked division of the bronchus, penetrates the lobule until it reaches a point beyond the terminal bronchus. Here it divides quite abruptly into as many branches as there are atria. These arteries do not come directly to the surface of the pleura and, as a rule, no artery passes to the periphery of the lobule.

The air-sacs lying within the proximal portion of the lobule receive a separate supply, small branches being given off just before the artery penetrates the lobule.

Within the lobule the artery spreads out on the central side of the air-sac, where the capillary network arises, and, after extending over all the air-cells, empties on the peripheral side into the veins.

The pulmonary veins, as has been stated, are found on the peripheral side of the air-sacs. The juxtaposition of artery, bronchus, and vein is strictly confined to the hilus; within the lung the artery accompanies the bronchus, but the vein keeps as far away from artery and bronchus as possible. The veins keep on the periphery of the lobule except in a few instances, when they send branches between the air-sacs; these branches collect the blood from the capillary networks on the atria.

There is one important exception to the rule that the artery is central and the vein peripheral. Arising one from either side of the extremity of the terminal bronchus, and receiving blood from the final ending of the bronchial artery and from adjoining air-sacs, are two short but good-sized veins which pass by the shortest route to the nearest venous trunks.

The same rule holds for a bronchus of the second order, with its branches: the artery is central and the veins peripheral, except, again, for two veins arising at the point where branches are given off.

The pulmonary veins receive blood from the following sources: (1) The capillary network arising from the terminations of the pulmonary artery on the central side of the air-sac; (2) the capillary network of the pleura; (3) the capillaries of the bronchial artery, both those which surround the terminal bronchi, and those which arise at the angles where branches are given off from a bronchus.

All the veins, except the small branches which arise at the terminal bronchi, are on the periphery of the lobule; the veins from the terminal bronchi are situated within the lobule.

The bronchial artery forms a vascular plexus in the walls of the bronchi. The blood conveyed by the branches of the bronchial artery is carried away by the pulmonary veins and not by the branches of the pulmonary artery. No branches of the bronchial artery are found on the distal side of the terminal bronchi (Miller).—Ed.]

Most of the **nerves** of the lungs originate from the anterior and posterior pulmonary plexuses; they are mixed vagus and sympathetic fibers. The vagus fibers belong to the musculature and the sympathetic to the blood-vessels. These latter come from the first thoracic ganglion. (Vulpian, "Leçons sur l'appareil vaso-moteur," 1875, t. II, p. 43.) On those fibers which belong to the bronchial wall, Remak described groups of ganglion cells. (Müller's "Archiv für Anatomie und Physiologie," 1844.)

Lymph-glands lie in large numbers behind, beside, and below the bronchi and directly take up the branches which come from the two bronchial networks, the mucous and the submucous. They are called the bronchial glands. Their number is considerable: besides numerous small ones, we can count over twenty which equal or exceed the size of a bean. They are distinguished from all the other glands of the body by the fact that they become gradually pigmented black; the pigmentation begins in early youth and increases with age. They take up principally the lymph-vessels of the lungs, but also the vessels of the bronchial mucous membrane and of the submucous lymphatic bronchial plexus. From the glands the vasa efferentia empty partly into the thoracic duct; in part they form a trunk known as the bronchomediastinal trunk, which leads independently into the left subclavian, and another which goes to the right subclavian vein; finally branches pass directly into the vena azygos.

The principal masses, consisting of from ten to fifteen glands of about the size of a cherry-pit, occupy the space between the right and left bronchi, principally below the bifurcation, and extend to the pericardium. Single glands surround the bronchi and lie on the pulmonary artery. The glands are relatively better developed in the child than in the adult. They receive their blood from the bronchial arteries and empty into the bronchial veins.

A description has been given by Teichmann * of the lymph-capillaries of the bronchi. He was able to show a network of lymph-vessels in the mucosa and a second in the submucosa. That of the mucosa has a direction parallel to the long axis of the bronchi, while that of the submucosa runs transversely, on the cartilaginous rings. The relation between lymph-vessels and blood-vessels is quite uniform; immediately beneath the epithelium the vascular capillaries are found, and under these lie the lymphatic capillaries. Teichmann has not investigated the lymph-vessels in the finer and finest bronchi. The assertion that free openings lead from the alveoli into the lymph-vessels of the lungs has not been confirmed and is very improbable.

[The lymphatics of the lung consist of a system of closed tubes situated in the walls of the bronchi, in the pleura, and along the branches of the pulmonary arteries and veins.

In the *bronchi* the lymphatics form a network with a somewhat long and narrow mesh; they diminish in size from the hilus to the terminal bronchi, where they end in three small vessels. Beyond the terminal bronchus no lymph-vessels are found in connection with the air-spaces; that is, the atria and air-sacs have no lymphatics in their walls. From this network of bronchial lymphatics, which lies exterior to the network of bronchial blood-vessels, branches pass:

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* "Das Saugadersystem," Leipzig, 1861.

1. To the pulmonary artery which accompanies the bronchus;
2. To the veins which arise from the angles where branches are given off; and,
3. To the veins which arise from the distal extremity of the terminal bronchus.

One of these three branches passes to the *artery* and accompanies it in its course; where the artery joins another to form a larger vessel the lymph-vessels also unite, forming a proportionately larger vessel. The large trunks of the pulmonary artery and vein are accompanied by two, or sometimes three, lymph-vessels connected by numerous loops, so that each blood-vessel is surrounded by a plexus of lymphatics. The smaller arteries and veins are accompanied by a single lymph-vessel which is situated between the artery and the bronchus.

The remaining two lymph-vessels arising from the terminal bronchus pass, one on either side, to the small *veins* which arise from this point; they usually have a more spiral course than those about the artery. Lymph-vessels also pass from the bronchus to the veins which are found at the point where a branch leaves the main bronchus. There is, therefore, at the point where bronchi branch, practically the same relation of lymph-vessels as at the terminal bronchus: three lymphatics, one of which passes to the artery, the other two to the veins. The small veins have but a single lymph-vessel accompanying them, but the larger veins have two, and rarely three, main vessels which are connected by loops and form a network with a long mesh about the veins.

Finally, a lymph-vessel accompanies the veins that pass to the pleura, and unites with a network of lymph-vessels found in the pleura.

The surface of the lung is covered by an extensive network of large lymph-vessels, in the meshes of which lies a network of finer vessels. The lymphatics of the *pleura* form at the hilus of the lung four or five large trunks which anastomose at this point with the lymphatics belonging to the lung.

Along the border of the lung the large lymph-vessels of the pleura often dip into the lung to some depth, only to come to the surface again after a short interval. These loops do not appear to give off any branches to the lymphatics of the lung. Valves are present in large numbers in the lymphatics of the pleura. There is an anastomosis between the lymphatics of the pleura and those of the lung; it is, however, confined to the hilus, no anastomosis taking place on the pleural surface.

The presence of stomata between the epithelial cells covering the pleura is questionable (Miller).—Ed.]

The relation of the pleura to the main bronchus is such that, at about the middle of its course, the union of the pulmonary with the mediastinal pleura, which extends from above toward the hilus of the lung, is closely crowded against the bronchial wall. The lower point of union, on the other hand, is at some distance from the bronchus. A schematic representation of these relations is found in the atlas of Heitzmann.

It is said that the entire volume of the bronchi amounts to 100 to 120 c.c., while that of all the respiratory tissue is estimated at over 3000 c.c.

In inspiration the air-pressure in the bronchi is negative, but in quiet respiration it scarcely falls below 2 mm. Hg; in expiration it is just as much positive; by rubbing and pressing one can run it up to over 80 mm. Thus one can in the bronchial tree cause very great variations of pressure. The bronchial muscles contract on irritation of the nasal mucous membrane, just as on excitation of the peripheral ends of the vagus; the reflex from the nasal mucous membrane on the bronchial muscles is brought about by way of the vagus. These facts are confirmed by numerous investigators.* An increase of the bronchial pressure is connected with this contraction of the bronchial muscles. In section of the vagus the bronchi of the same side usually relax surprisingly (dog).

The experiments of Riegel and Edinger showed also that excitation of the vagus *in continuo* was always followed by a marked inflation of the lungs, and, if continued, by a considerable depression of the border of the lungs, although the air-movements were slight. Excitation of the peripheral end of the vagus never caused inflation of the lungs; this was caused only by irritation of the central end. It consisted of a reflex of the vagus on the phrenic, for after its section the effect was wanting.

The sympathetic, on the other hand, does not influence the bronchial musculature. It contains two kinds of fibers: first, those which with weak excitation make the breathing slower and deeper; second, those which on weak excitation make the breathing quicker and more superficial. On strong excitation of both groups of fibers the respiration stops. In the rabbit these fibers enter the spinal cord between the fourth and fifth thoracic vertebræ.† In inspiration the bronchi are relatively narrow, their diameter being shortened as much as the cartilage plates will permit. In expiration there is scarcely any change, if the breathing is done quietly with the glottis open; but if the glottis is narrowed, or the pressure which the expiratory muscles can exert is brought into play (for the respiratory muscles are not used by the healthy normal individual), both the trachea and bronchi are widened.

The **mucous membrane of the bronchi** acts like that of the trachea: it secretes mucus, derived partly from the mucous glands and partly from goblet cells. I do not venture to say how the secretion of the one differs from that of the other. Panizza believes that the glands have a more fluid secretion, while that of the cells is more viscous. The secretion does not form a homogeneous layer, but cell-like balls filled with myelin forms; particles of dust and soot are found in a ground substance which has a striated appearance. Hence myelin cells and pigment cells have long since been distinguished in the secretion.

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* Of these, I name only Brown, Einthoren, and Lazarus. Literature is given in Riegel and Edinger, *Zeitschr. f. klin. Medicin*, v.

† Hamburger, *Med. Centralblatt*, 1891, S. 45.

The pigment cells are easily distinguished by their pigmented contents; they are connected by successive transitional stages with others that are quite structureless, have no nuclei, and only slightly granular contents; and with yet others that contain true myelin forms.

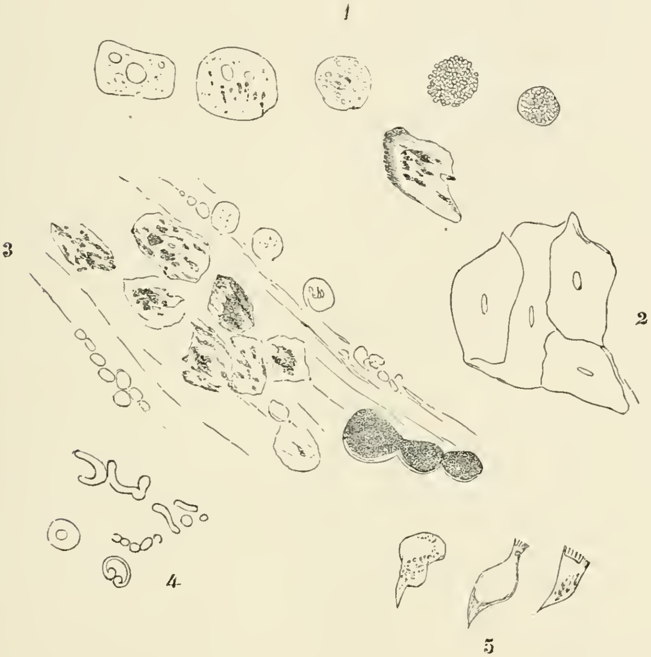

FIG. 3.—1 and 3, Various cell forms in the sputum of a healthy man; fatty, partly degenerated cells; pigment cells; cells of uncertain character; altered leucocytes. In 3 the usual group of changed, pigmented alveolar epithelium. 2, Normal alveolar epithelium, very rare, the nucleus visible only on staining. 4, Myelin forms. 5, Desquamated, changed bronchial epithelium; very rare.

These myelin forms are characterized by their strong refraction of light and by the sharply contoured, peculiar shapes which they show. They may lie in small numbers in the cell-like forms, they may fill these completely, or they may be met floating freely. They may then imitate spore forms or they may even appear like more or less modified spermatozoa.

These myelin and pigment cells are now generally regarded as exfoliated and modified alveolar epithelium. Smith and Guttmann assert this with a good deal of positiveness, and endeavor to show transitional forms from the one to the other. What decides me to concur in their opinion is the fact that these cells are undoubtedly found in the alveoli in the finest sections of well-hardened lung. In places they are found in such positions that they cannot be regarded as anything but epithelial cells *in situ*. Although they already begin to change their habitus and to take on pigment, yet they still have their characteristic nuclei and lie fitted into the mosaic of other cells. It is very difficult to understand how they reach the bronchi; judging by the behavior of inhaled coal-dust one would think that these epithelial cells would be much more likely to reach the lymph-passages than the bronchi. The opinion of Panizza that peculiar motile drops of mucus separate themselves from the mucous membrane, and that these take up the dust lying on them and pass outward, has therefore much to recommend it. But it cannot, without violence, be brought into harmony with the above-mentioned findings, and I have therefore given it up.

It is not always very easy to explain the origin of the morning sputum of quite healthy people, which is expectorated in the form of larger and smaller masses (up to the size of a cherry-stone). These masses are chiefly characterized by their gray color and tough, elastic, consistency. The larger masses appear distinctly compounded of the smaller ones. They can be hardened, sectioned, and stained, and then present very interesting pictures. In part we find a hyaline, slightly fibrous portion, in part a very cloudy, densely granular one, and between them we find the pigment and myelin cells in groups and rows.

Many cells are undoubtedly present which have wandered out from the blood-vessels; they are, however, so changed that we can see only circular or, if they are crowded together, angular forms, with pale contents which are not at all characteristic. It is difficult to recognize them as real cells; but nuclei or the rudiments of such can be demonstrated after treating the cells with liquor potassæ.

In favorable dry preparations a sufficiently useful stain may be secured with the triacid, which shows that these cells act quite like polynuclear white blood-cells, and this in a certain stage of degeneration I consider them to be. They do not usually contain any pigment and only take it up in exceptional cases, and I could never quite convince myself of its presence. They form, however, such masses with the pigment cells that they easily have the appearance of pigment cells. Ciliated cells or their recognizable remains I have rarely seen in the normal sputum, and hence regard them as exceptional.

Dr. Baruban * has reported on the bronchial epithelium of a healthy man, who, having been sentenced to death, smoked an extraordinary number of cigarettes in the last hour before his execution. He found pieces and strips of pavement epithelium among the ciliated cells. In the finer bronchi the loss of the cilia and the strong pigmentation were surprising. True catarrh was not present.

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* According to a reference in the supplement to the *British Medical Journal*, October, 1890.

MALFORMATIONS OF THE BRONCHI.

1. Compare trifurcation of the trachea.

2. Chiari* reports a congenital diverticulum in the right bronchial trunk, which was as large as a bean and was regarded by him as an accessory branch of this bronchus, which had remained rudimentary.

3. Chiari also reports the presence of *two* eparterial bronchi from the right bronchial trunk, the lower of which originated at the normal place of exit of the eparterial bronchus, while the upper was considerably higher, near the point of division of the trachea. The ordinary eparterial bronchus has, on the right side, normally three branches—a dorsal, a ventral, and an ascending. The latter may be partly or wholly replaced by a special eparterial bronchus. The point of origin of this varies, being at times near the bifurcation, at other times nearer the normal eparterial bronchus; it is, as already remarked, variable in its location, and may even originate as far back as the trachea, and then constitutes the tracheal bronchus, thus explaining the trifurcation of the trachea.

4. A very remarkable observation is that of Ratjen.† In a man who died of an affection of the brain at the age of forty-nine, the left bronchus ended blindly an inch below the bifurcation; its continuation formed a strand of connective tissue of about the thickness of a penholder, about one and a half inches long, which then passed again into the continuation of the bronchus, leading into the left lung. The latter was entirely atelectatic, very small, gray, and pigmented. The bronchi contained thickened mucus with lime and fat-molecules. Ratjen considers this case a congenital malformation. The presence of the pigment in the lung makes this, however, very improbable.

INJURIES OF THE BRONCHI.

LITERATURE.

Gosselin: "Mémoires de la Société de chirurgie de Paris," 1847.—Bardeleben: "Lehrbuch der Chirurgie und Operationslehre," Bd. III, S. 599, 1875.—Courtois: "Etude sur les contusions du poumon sans fractures des côtes," Thèse de Paris, 1873, No. 180.

Under this head we will consider only a part of the injuries to the larger bronchi. The injuries of the smaller bronchi belong to those of the lungs. The injuries which are due to the fact that foreign bodies have passed through the trachea to the bronchi are treated under the head of foreign bodies. The rest may now be divided into direct and indirect. For the presence of the former the opening of the chest-wall is a necessary precedent; the injuring force must penetrate to the bronchus—as a stab or a shot. These injuries are discussed in works on surgery in connection with perforating wounds of the chest. I do not wish, however, to pass over the *indirect* injuries.

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* *Prager med. Wochenschrift*, 1890, Nr. 46.

† *Virchow's Archiv*, Bd. XXXVIII, S. 173, 1867.

Here the thorax suffers only a compression, and yet a tearing of the tissues which it protects occurs. How this happens has been the occasion of much discussion. The controversy was terminated by the exposition of Gosselin and Bardeleben, who showed that the presence of air, that at last cannot on account of the compression escape from the lung in the normal way, is necessary to produce laceration of the tissues. This was shown also by Courtois, by means of experiments especially directed to this purpose. The air cannot escape if, for instance, the glottis is closed at the moment of the injury.

What usually happens in such contusions is a tearing of the lung tissue; and, although the fine bronchial twigs must necessarily be injured at the same time, that subject will not be treated under the diseases of the bronchi.

In this place we have to deal only with cases of injury to the bronchi in which the lungs are relatively little or not at all involved. It is clear that such cases can include only injuries of the large bronchi. The number of these cases is very small; generally the ribs are broken at the same time, and then the injury is ascribed to the splinter which penetrated into the thoracic cavity from the broken rib. The observations known to me are as follow:

Carbonnell, 1865, "Bullet. Soc. anatom. de Paris," II. Série, t. x, pag. 17, vol. 40. The right bronchus was separated from the trachea at the point of bifurcation.

Seuvre, 1873, "Bullet. Soc. anatom. de Paris," III. Série, t. VIII, vol. 48. Run over by an omnibus. The right bronchus was entirely separated from the trachea.

Baader, 1877, "Correspondenzblatt für schweizerische Aerzte," S. 653. Run over by a heavy wagon. The left bronchus was torn by a sharp splinter.

Sheld, 1889, "Transactions Path. Soc.," vol. XL, p. 38. Complete tearing of the bronchus at the bifurcation by a wagon-pole. Third and fourth ribs were broken.

In the following cases it is to be assumed that the bronchial injury was not determined by the injury to the thorax:

Broca, "Progrès médical," 7. Febr. 1885, pag. 108. Crushing. Many ribs are broken with tearing of the lungs. Left-sided pneumothorax through rupture of the left bronchus. In the posterior portion of the left bronchus, a millimeter before its entrance into the lung, is a perforation the size of a bean. It is at the place where the pleura bends around, and here the air escaped into the pleura.

Knoepfler, "Des ruptures bronchiques," Nancy, 1886, pag. 92. Crushing. Fracture of the first left rib, with luxation of the left clavicle. Fracture of the third and fourth ribs on the right side. Rupture of the left bronchus, left-sided pneumothorax. Death. If air is blown into the trachea, it is seen passing out into the left pleural cavity through a small opening on the posterior surface of the left bronchus, shaped like a flute-stop. The bronchus is torn between its second and third rings, immediately outside of the place where the arch passes over it. The two fragments are united only by a bridge of fibrous tissues to the posterior wall of the bronchus, which is also torn lengthwise.

In the case of Ilott,* a six-year-old boy was run over in such a way that the thoracic walls showed no fracture at all and no injury; yet the lung tissue was torn, there was pneumothorax and considerable hemorrhage; the left bronchus was torn across at the first division. Death half an hour after the accident.

According to the location of the injury the air may pass out into the mediastinum or into the pleura. There is always abundant blood extravasation. The course has been until now always fatal, but the opening may be very small, and so a fortunate outcome might be considered possible.

FOREIGN BODIES IN THE BRONCHI.

LITERATURE.

Porter: "Observations on the Surgical Pathology of the Larynx and Trachea," London, 1837, pp. 109–230. (Experiments on dogs. A small piece of radish introduced through a wound in the trachea was repeatedly expelled by coughing, though the foreign body was pushed into one of the bronchi.)—Stokes: "Die Brustkrankheiten von Behrens," Leipzig, 1844.—Hager: "Die fremden Körper im Menschen," Wien, 1844.—Gross, S. D.: "A Practical Treatise on Foreign Bodies in the Air-passages," Philadelphia, 1854.—Kühn, in Günther's "Lehre von den blutigen Operationen," v, 1864.—Bourdillat: "Gaz. méd. de Paris," 1868, 7, 9, 10, 13, 15.—Preobraschensky: "Wiener Klinik," 1893, Nr. 8–10.—Kobler: "Wiener klin. Rundschau," 1895.—Willard, De Forest: "Removal of Foreign Bodies from the Air-passages," "Jour. Amer. Med. Assoc.," October 26, 1901.—Godlee, R. J.: "On the Effects Produced by Foreign Bodies in the Bronchial Tubes," "R. Med.-Chir. Soc. Trans.," vol. LXXIV, 1896.—Wood, Geo. B.: "Foreign Bodies in Air-passages," "Phila. Med. Jour.," 1879.—Weist, J. R.: "Foreign Bodies in Air-passages," "Trans. Am. Surg. Ass.," 1882.

It is not so very rare an occurrence for foreign bodies to reach the bronchi through the natural passages. What is possible in the way of accidents is shown by the following table, which represents the best-known and most important observations afforded by the literature. A close examination of these special cases is needed to gain an understanding of the difficulties that the physician has to cope with in certain cases. A complete collection of all that has been reported on this subject is not the object of this work; there are numerous unsatisfactory observations, which might, however, be utilized in some way. Preobraschensky has given a very complete analysis of the clinical material at hand

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* *Brit. Med. Jour.*, Sept., 1890, p. 733.

In spite of the large number of observed cases, we see how much yet remains to be done and how great is the lack of exact observations. One should not be misled by writers who assert that it is useless to collect further cases. It is only useless to collect poorly observed and poorly described cases.

Foreign bodies act differently according to their kind and size. It is therefore not right to treat them all alike. It is necessary to classify, and I have therefore made five classes in order, as far as possible, to group together bodies having the same physical characteristics and presumably characterized by similar symptoms and a similar course:

1. Smooth, round pieces of metal and glass (coins and bullets).
2. Hard, irregular, sharp, and pointed bodies (needles and splinters of bone).
3. Soft, rough, bodies (heads of grain).
4. Bodies which swell on soaking (fruit-seeds). Hard fruit-seeds especially.
5. All other foreign bodies.

As in the case of foreign bodies in other regions, children furnish the largest number of examples; but the percentage of adults is also quite large. Anything that a man has in his mouth may at any time, by an accidental reflex inspiration, be drawn into the bronchi. The bad habit of putting needles and other similar objects into one's mouth instead of laying them down cannot, therefore, be too severely condemned. The accident is most likely to occur when sensibility and reflex activity are diminished; for the muscles of the glottis, which contract on the slightest irritation, are the sentinels who by their sensibility and motility guard the body in ordinary life against all intruders. Chloroform narcosis is therefore a factor that favors the entrance of foreign bodies; it has repeatedly happened that a tooth extracted during anesthesia has slipped down into the trachea. Drunkenness and all other forms of intoxication, as well as natural sleep, diminish the vigilance of the sentinels that stand guard over the glottis. In diseases of the brain they may be rendered completely powerless, and it is well known how often people who swallow badly die from the aspiration of foreign bodies.

As a rule, the aspirated foreign body, after it has once passed the glottis, proceeds at once to a point in the bronchial system which corresponds to its size, and there becomes firmly fixed. More rarely the foreign body remains for a time in the trachea, and later passes down into a bronchus. If it strikes the spur at the bifurcation, or at any other point where a branch is given off, it may be thrown back by the fit of coughing which at once follows, and this movement may be repeated many times before the foreign body finally lodges in a bronchus. It rarely happens, also, that the body first lodges in the trachea and later passes down into the bronchus.

SMOOTH, ROUND PIECES OF METAL OR GLASS. (COINS, BULLETS.)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Young, "Lancet," II, 1879, S. J.* 188, p. 290, 1880.	Boy of five years.	Oval glass body in the right bronchus.		One hundred and two days. Immediate tracheotomy without result. Subsequently coughed up.	Recovery.
Bell, 1881, "Edinb. Med. Jour.," S. J. B. 198, p. 171.	Man.	Sixpenny piece in the left bronchus.		One day. Patient inverted and tapped on the back at the spot corresponding to left bronchus.	Recovery.
Keeling, 1883, "Lancet," S. J. B. 204, p. 188.	Boy of three and a half years.	A glass bead in the left bronchus.	Coughed up.	Three days. Tracheotomy at first brought no relief; on the third day the bead was coughed up.	Recovery.
Brodie, "Med-chir. Trans.," B. 26.	Adult male.	A half-sovereign.	The piece remained movable. The patient could cause an upward or downward movement by changing his position, and in other ways.	Tracheotomy on the twenty-fourth day without result. Several days later the patient's head was lowered and the piece was suddenly cast out.	Recovery.
Sprengel, "Centralbl. für Chir.," 1891, No. 14.	Girl of five years.	Glass bead.	Severe dyspnea, with inspiratory retraction on both sides. Respiratory murmur on the left very indistinct. The bead was lodged at the bifurcation of the trachea. The air could pass through the hole in the bead to the right bronchus, while the body of the bead obstructed the passage to the left bronchus.	Tracheotomy on the seventh day.	Recovery.

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*S. J., in the following tables, means "Schmidt's Jahrbücher."

SMOOTH, ROUND PIECES OF METAL OR GLASS. (COINS, BULLETS.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION.)	SYMPTOMS.	TREATMENT.	RESULT.
Cork, quoted by Sander, p. 340.	Man, thirty-five years old.	Sixpenny piece in the right bronchus.	After some days the coin was expelled during sleep.	No treatment.	Recovery.
Lescure, "Mem. d. la Soc. Roy. d. Chir.," vol. 351, Paris, 1819.		A *Louis d'or.*	The right lung nearly destroyed by suppuration.	No treatment.	Death.
St.-Germain, "Arch. des maladies de l'oreille," etc., II.	Child, eight years old.	Bead in the left bronchus.		Tracheotomy.	Recovery.
Maurder, "Lancet," II, 1876, p. 854.	Boy, thirteen years old.	Glass button in the left bronchus.	April 23d: In good condition. A peculiar, sibilant cough. May 11th: Fever, respiratory murmur is now present posteriorly on the left side; very weak at the base.	Tracheotomy. Introduction of a silver wire and removal of the object.	Recovery.
Niemeyer, "Therapeut. Monatshefte," 1891, No. 38.	Young man.	Rifle bullet in the left bronchus.	Pain in the second right intercostal space, a finger's-breadth to the right of the edge of the sternum.	Patient was laid on a couch, with head and chest hanging over, so that the head rested on the floor.	Bullet was coughed up. Recovery.
Naismith, "Brit. Med. Jour.," June, 1887, p. 1276.	Boy of seven years	Glass bead in trachea and bronchus.	The observation is of especial interest, as one could with great certainty diagnose different positions of the bead; at one time it would roll about in the trachea; then it obstructed the left bronchus, and afterward the right bronchus. During obstruction of the bronchus, the respiration increased to 32.	Tracheotomy. Three days later the bead was coughed up.	Recovery.
Page, "Brit. Med. Jour.," July, 1889, p. 13.	Boy of nine years.	Smooth carnelian stone, 1.6 cm. long, 1.3 cm. wide, 0.6 cm. thick.	One could hear, at about the middle of the second right costal cartilage, a peculiar rattling or blowing sound, as if air were passing through a narrow tube.	Tracheotomy on 11th day. Great difficulty in causing movement of stone was noticeable. It was 13.5 cm. below tracheal opening.	Recovery.

Fergusson and Johnson, "Lancet," October, 1878.	Boy.	Rather long glass bead in the left bronchus.		No interference.	After some months the bead was coughed up. Recovery.

HARD, IRREGULAR, SHARP AND POINTED OBJECTS. (NEEDLE AND BONE SPLINTERS.)

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Achard, "Frorieps Notizen," vol. 5, p. 302.	Man, fifty-six years old.	Two small bones.	Fever and purulent expectoration, continuing for three months after the bones were coughed up.	No treatment.	Recovery.
Stokes, *l. c.*	Adult female.	Vertebra of a pigeon.	Pain, hemoptysis, hectic symptoms. The bone was coughed up after seventeen years.	No treatment.	Death one and a half years after the discharge of bone.
Howship, quoted by Stokes.		Iron nail in the right lung.	Painful cough. Pain localized at one point in the right lung. Repeated attacks of hemoptysis. The object was coughed up.	No treatment.	Partial recovery.
Orth, quoted by Capesser.	Girl.	Piece of bone in a branch of the left bronchus.		Tracheotomy without result.	Death after two months.
Cork, cited by Sander, p. 340.	Young man.	Projectile of blow-pipe (nail with tuft of cotton).	After two months, it was coughed up with a large amount of pus.	No treatment.	Recovery.
Carpenter.	Man.	Four false teeth on a silver clamp.	No symptoms. Thirteen years later fever and pleuritis lasting nine months. Old fistulous opening in the right lung.	No treatment.	Death.

HARD, IRREGULAR, SHARP AND POINTED OBJECTS. (NEEDLE AND BONE SPLINTERS.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Curpani, Morgagni, February, 1888.	Woman of fifty-one years.	Lance-shaped piece of bone.	The object was coughed up.		Recovery.
Settegast, "Archiv für klin. Chir.," vol. 23, p. 272.	Workwoman; age twenty-one years.	Tracheal tube, which she had worn for years, because of tracheotomy after typhoid, slipped into left bronchus.	Fearfully torturing cough. After four weeks, tube was coughed up and expelled through the trachial wound.		Recovery.
Fredet, cited by Sander, "Gaz. hôp.," 1865.	Child of two years.	Ear-ring in right bronchus.	Nothing was known of the presence of the foreign body during life. Pneumonia on the right side and bronchopneumonic areas on the left side.		Death.
Monckton, "Brit. Med. Jour."	Boy of seven years.	Nutshell in the lungs.	Expectorated after twenty-three weeks.		Recovery.
Finkelburg, "Virch. Arch.," vol. 19	Man, thirty-four years old.	Piece of bone in right bronchus.	Fever, pneumonia, and fetid expectoration.	No treatment.	Death after forty-four days.
Page, "Brit. Med. Jour."	Child, three years old.	Slate pencil in left bronchus.	Pneumonia on both sides. Long sickness.	No treatment.	Death a year later after a sudden fit of choking.
Herrich, cited by Sander.	Boy, fourteen years old.	Needle in right lung.	Herrich, being called four years later, because the boy had fever, found pneumonia. Needle was coughed up.		Recovery.
Lesselier, Obs. 67, cited by Poulalion.	Man.	Splinter of bone.	Symptoms of phthisis. One day he suddenly coughed up the splinter of bone.		Recovery.

Hutchinson, "Trans. of Pathol. Society," vol. 13, 1862.	Man of sixty years.	Splinter of bone.	Attacked at a dinner by a severe fit of coughing. Developed a condition resembling phthisis, with frequent attacks of hemoptysis, and lasting several months. Finally the splinter was expectorated with a large amount of pus.		Recovery.
Cheadel and Smith, "Brit. Med. Jour.," 1887, Jan. 14, p. 76.	Girl of nine years.	A metallic lead-pencil case.	Dulness and weakened breathing on the left. No dyspnea. Occasional cough. The foreign body remained for fourteen days without producing any marked symptoms.	Tracheotomy. The object was withdrawn with forceps.	Recovery.
R. M. Simon, "Brit. Med. Jour.," Nov., 1887, p. 1153.	Boy of six years.	A checker in the right bronchus.	Stridulous breathing and dyspnea. A few rhonchi on the right side; later, absolute dulness, and absence of respiratory murmur. On the day after operation, the dulness was gone.	Tracheotomy. An oiled feather was introduced and then the object was coughed up.	Recovery.
Fischer, "Corresp. für Schweizer Aerzte," 1893.	Man of twenty-six years.	A piece of bone 1½ cm. long.	Moist râles in the right lower lobe. Pain under the clavicle. After some time, marked expiratory dyspnea. Cachexia. Finally bone was coughed up with much pus.		Recovery.
Hughes, 1815, S. J. T. 85, p. 243.	Man of thirty-eight years.	A fish-bone, 2 cm. long, in the posterior, upper portion of the left lung.	Abscess formation.	Fifteen weeks.	Death.
Struthers, 1852, S. J. T. 87, p. 244.	Man of twenty-two years.	Piece of bone firmly lodged in the right bronchus.	Phthisical hemorrhages.		Died after two and a half years.
Oppolzer, 1853, S. J. T. 80, p. 206.	Male, sixteen years old.	A rhomboid piece of bone as large as a bean in the main bronchus of the left upper lobe.	Bronchiectasis. Brain abscess in the right posterior and in the left middle and posterior lobes.	No treatment.	Died after three years.

HARD, IRREGULAR, SHARP AND POINTED OBJECTS. (NEEDLE AND BONE SPLINTERS.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Theile, "Deutsche Klinik," 1853, No. 17.	Child.	Small piece of charcoal in the left main bronchus.	Pneumonia on the left side. Small, symmetric cavities scattered through the whole lung.		Died after fourteen days.
Capesser, Diss. Geissen, 1852.	Girl of twenty years.	Piece of a molar tooth in bronchus.			Died after one month.
Abercrombie, 1821, S. J. B. 7, p. 177.	Woman.	Tooth.	It was coughed up after two years and seven months.	No treatment.	Died about a year later from the sequels of the pulmonary affection.
G. D. Angelis.	Girl.	Whistle, 3 cm. long and 70 mm. in diameter, in the right bronchus.	At each inspiration and expiration the whistle gave out a sound which could be heard at a distance of 15 meters (50 feet).		
Foster. (?)		Small whistle in the right bronchus.	In breathing there was a whistling sound, which became louder after coughing.	Tracheotomy and extraction of the whistle.	Recovery.
Hecker, "Wochenschr.," 1885, No. 8.	Woman of sixty years.	Piece of cartilage in the right bronchus.	Loss of consciousness, without paralysis; right side immovable, although breathing greatly accelerated.	Emetics.	Recovery.
Pick, cited by Kobler, "Semon's Journal."		Pipe stem so wedged into the bronchus that the patient breathed through it.			

Israel, "Arch. für klin. Chir.," vol. 34.		Small piece of tooth infected with actinomyces.			Death from actinomycosis of lungs.
Kobler, "Wiener klin. Wochenschr.," 1889, No. 33.		Piece of bone.	No pain at first.		Death.
Boyce, "Lancet," 1887.		Mouthpiece of a pipe.	It was expectorated two months later, after the patient had swallowed a fish-bone.		
Weil, "Deutsches Arch. für klin. Medicin," vol. 14, p 94.	Boy, thirteen years old.	Shell of hazelnut in the right bronchus.	Peculiar, gasping murmur, an expiratory whirr on the right side of the chest in breathing. No pain, no cough. Expectorated.	No treatment.	Recovery after three and a half months.
Sander, "Deutsches Archiv für klin. Medicin," vol. 16, p. 330.	Man of thirty-eight years.	Piece of bone in right lung.	A very unpleasant sensation of pressure at an indefinite place in the right chest. At times feeling as if bone moved up and down. Coughed up after ten weeks.	No treatment.	Recovery.
Sander, "Deutsches Archiv fur klin. Medicin," vol. 16, p. 330.	Girl of ten years.	Shoe-nail.	Fever with purulent expectoration. No pain. Coughed up after nineteen weeks.	No treatment.	Recovery.
Sander, *l. c.*, p. 339.	Girl of twenty years.	A piece of tooth during extraction fell into right bronchus.	Pneumonia on right side in lower part. Later dyspnea. Delirium at night. Coughed up after one month.		Death immediately afterward.
Vierhuf, "St. Petersburger med. Wochenschr.," 1889; "Med. Centralbl.," 1889.	Child of seven years.	Small piece of wood.	Catarrh, pneumonia. After some retching, it was vomited up with pus.		Recovery after twenty-six days.
Landé, "Deutsche Vierteljahrschr.," XIII, 1873.	Boy of seven years.	A molar, while being extracted, fell into left bronchus.		Tracheotomy after some hours.	Recovery.

HARD, IRREGULAR, SHARP AND POINTED OBJECTS. (NEEDLE AND BONE SPLINTERS.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Laidler, "Jahrbuch für Kinderkrankheiten," N. F., vol. 13.	Boy.	Metal penholder 1½ inches long in the left bronchus.		Tracheotomy.	Recovery.
Gross, "Practical Tract on Foreign Bodies in Air-passages."		Plate with four teeth.	Pieces remain in bronchus.		
MacCormac, "Brit. Med. Jour.," 1886. Jänner, p. 70.	Girl, twenty-five years old.	During the extraction of a tooth, a piece of the forceps broke off and fell into the right bronchus.	Pain on the right side, beside the sternum, corresponding to the second right intercostal space.	Extraction. The piece was in the bronchus 4 cm. behind the bifurcation.	Recovery.
Voltolini, "Monatsschr. für Ohrenheilkunde," 1879.	Man.	Piece of polyp-forceps in right bronchus.	Coughed up after nine months.		Recovery.
Marsheim, "Deutsche med. Wochenschr.," 1878.	Man of forty-two years.	Piece of bone in right bronchus.	Coughed up after four months.		Recovery.
Volland, "Deutsche med. Wochenschr.," 1878.	Man.	Cervical vertebra of a chicken in right bronchus.	Putrid bronchitis on the right side.	Over one and a half years.	Died from the putrid bronchitis.
Chambers, 1881, S. J. B. 198, p. 171.	Woman of thirty-five years.	Tooth in the left bronchus.	Coughed up.	Six days.	Recovery.
Langier, 1881, "Prager med. Wochenschr.," S. J. B. 198, p. 171.	Man.	A small stone.	Coughed up.	One month.	Recovery.

Harris, 1882, "Med. Times and Gaz.," S. J. B. 199, p. 49.	Boy of twelve years.	Metal needle 1½ inches long in right bronchus.	The patient felt the needle move up and down in the right side of the chest. Coughed up.	Twenty-five days.	Recovery.
Landgraf, "Deutsche med. Wochenschr.," 1890, p. 43.	Man of forty-six years.	Piece of cricoid cartilage in the right bronchus after syphilis.		Diagnosis not made. Tracheotomy on account of the attacks of dyspnea; no relief.	Death.
Schrötter, "Vorlesung über Kehlkopf krankheiten."		Piece of bone, at first wedged in the larynx below the true vocal cords, finally fell into right bronchus.			Death.
Schrötter, "Monatsschr. für Ohrenheilkunde," x, 1876, S. J. B. 181, p. 58.	Man of forty-five years.	Piece of bone in right bronchus just behind bifurcation.		Not diagnosed.	Death.
Ewald, 1878, S. J. B. 181, p. 59.	Child of six years.	Shawl-pin in left bronchus.	Constant whistling sound during whole time of expiration.	Remained thirty-three days. Tracheotomy.	Recovery.
Cusson, quoted from Kühn, 1843.	Child of six years.	Pebble.		Extracted from the main bronchus with curved polyp-forceps.	Recovery.
Langenbeck, quoted from Kühn, 1846.	Girl of twenty-two years.	Shell of hazelnut in left bronchus.	Pneumonia after four weeks.	On the fourth day, tracheotomy without result.	Death.
Forbes, Schm. J., vol. 75, p. 76, and vol. 69, p. 392.	Man of forty-six years.	Piece of bone at entrance to third branch of right bronchus.		No operation.	Died after eight weeks.
Gilroy, quoted from Kühn, 1826, S. J. B. 75.	Woman of forty years.	Small chicken bone in right bronchus.	Profuse expectoration with hectic fever.	Twelve weeks.	Death.

Hard, Irregular, Sharp and Pointed Objects. (Needle and Bone Splinters.)—(*Continued.*)

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Gottschalk und Schneevogt, cited from Kühn, 1852, S. J. 80, p. 85.	Man of fifty years.	Piece of bone 2 cm. long, $1\frac{1}{2}$ cm. wide, in the right bronchus.		Ten months. No operation.	Death.
Huston, cited from Kühn, 1830, Doublin J. F., 1834.	Girl of twenty-seven years.	Molar tooth loose in right bronchus.	Pneumonia and pleuritis.	Eleven days.	Death.
Weninger, Suppl. "Brit. Med Jour.," Oct., 1891.	Boy of thirteen years.	A nail with tin head (studding nail) entered the left bronchus. A few attacks of asphyxia, followed by disappearance of symptoms.	A year later he came to Dr. Weninger, with slight irritative cough and an area of dulness, 5 cm. in diameter, to the left of the sternum at the level of the bifurcation. At this point percussion was painful; six months later, nail was expectorated.		Improved. Recovered (?).
Mandowski, "Deutsche med. Wochenschr.," 1895, No. 30.	Girl of three years.	Aspirated a sewing needle 6 cm. in length the 19th of December. It lodged first in the trachea, but later reached the left bronchus.	Remained in fair health until the 10th of February of the following year. Then a chronic, caseous pneumonia developed, and later she had tuberculosis.	No active treatment.	Died on the 10th of April.
Gaethgens, "Allgem. med. Centralzeitung," 1879.	Girl of nineteen years.	Piece of almond shell, 3.5 cm. long, 2.5 cm. wide, in the right lung.	Pain on the right side of chest. Pneumonic symptoms.	No operation. Object was coughed up after about four months.	Recovery.

4	Rokitansky, "Lehrb. der pathol. Anatomie," III, 1861.	Boy.	Bolt of a blow-pipe in the beginning of the left bronchus.	During the attacks of coughing, it had penetrated the opposite wall of the trachea and pierced the adjacent truncus anonymus. Bronchitis, especially on the left side, and dark reddish-brown hepatization of the left lower lobe.	No operation.	Death from hemorrhage on the twelfth day.
	Pieniazek, "Wiener med. Blätter," 1888.		Blow-pipe bolt, armed with a pin, in the right bronchus.		Tracheotomy. Extracted with forceps which had to be introduced to a depth of 20 cm.	Recovery.
	Benthall, "Brit. Med. Jour.," September, 1882, p. 631.		Blow-pipe arrow, made with a needle and tuft of yarn, in the right bronchus.	Violent fits of coughing at times. After seven months, the needle rusted through and the arrow was expelled in two fits of coughing.	No operation.	Condition appears good. Result remains in doubt.
49	Jeffreys, "Brit. Med. Jour.," Dec., 1887, p. 1282.	Man of sixty-two years.	Piece of bone in the right bronchus.		No operation. Bone was coughed up.	Recovery.
	Hawkes, "Brit. Med. Jour.," Dec., 1887, p. 1383.	Boy of one year and eleven months.	Choked while eating part of a rabbit.	A vertebra of the rabbit was found in the bronchus supplying the upper part of the left lung. The lung was collapsed and in a condition of croupous inflammation.		Died on the tenth day.
	Finlay, "Brit. Med. Jour.," Oct., 1888, p. 807.	Boy, ten years old.	A piece of bone got into the right lung without exciting the suspicion of any one. Diagnosis was not made.	Treatment for marked bronchiectasis with putrid expectoration.	The bronchiectasis was opened by an incision. Pleura not adherent. Empyema and hemorrhage.	Death, fourteen days after the operation.

HARD, IRREGULAR, SHARP AND POINTED OBJECTS. (NEEDLE AND BONE SPLINTERS.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Kocher, "Wiener klin. Wochenschr.," 1890, No. 7–9.	Child of four years.	Broken point of an oil-burette, 3 cm. long, 5 to 6 mm. thick.	After two and a half months respiratory sounds were weakened on the right. Dulness in lower part.	Tracheotomy. Extraction. Forceps had to pass down 9 or 10 cm.	Death from abscess of brain in the right hemisphere, on twelfth day after operation.
Keferstein, "Therapeut. Monatshefte," 1890.	Woman of fifty-eight years.	Piece of bone. In trachea or bronchus.	Pain in middle of sternum. Convulsive coughing with severe dyspneic attacks.	Strong decoction of senega.	Recovery.
Lukas, "Brit. Med. Jour.," November, 1881, p. 818.	Boy of four years.	Small piece of gravel the size of a cherry pit. Movable. In trachea or bronchus.	Severe fits of choking.	Tracheotomy. Extraction.	Recovery.
Laver, "Brit. Med. Jour.," 1878, I, p. 71.		Piece of glass in the right bronchus.		Tracheotomy without favorable result.	Death one month later.

SOFT, ROUGH BODIES. (HEADS OF GRAIN.)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Bugh, "Jahrb. für Kinderheilkunde," N. F. 34.	Boy of five years.	Head of wheat in the anterior bronchial branch.		No treatment.	Death from gangrene of lung.

Slanski, "Gaz. med. d. Paris," 1837. Oppenheim, "Zeitschr.," vol. 6, p. 242.	Girl of twenty years.	Head of wild oats.	Abscess formation in right lower lobe of lung.	No treatment.	Death after eight months.
Bally, "Froriep's Notizen," vol. 10, p. 247.	Man of nineteen years.	Head of grain.	Abscess between seventh and eighth ribs.	No treatment.	Recovery.
Behrendt, 1832, S. J. B. 18, p. 89.	Boy of eleven years.	Head of grain.	Coughed up after three years.	No treatment.	Recovery.
Carrière, cited from Kühn, 1792.	Adult.	Head of grain.	Formation of abscess, from which the body was expelled.	No treatment.	Recovery.
Desranges, "Froriep's Notizen," 49, p. 252.	Boy of three or four years.	Head of grain.	Abscess in region of right kidney, from which the body was expelled.	No treatment.	Recovery.
Latz, S. J. B. 80, p. 396.		Head of grain.		Removed from an abscess.	Recovery.
Pincent, cited from Kühn, p. 176.	Boy of one year.	Head of grain.	Abscess between third and fourth false ribs.		Recovery.
Schmidt, "Hufeland's J.," 1833.	Girl of thirteen years.	Head of grain.	After thoracic disease lasting seven and one half years, the object was coughed up.		Recovery.
Vigla, "Jahrb. für Kinderkrankheiten," I, p. 295: cited from Kühn.	Boy of six months.	Blade of grass.	Abscess at fifth rib.		Recovery.
Vigla, "Jahrb. für Kinderkrankheiten," I, p. 295; cited from Kühn.	Girl of one year.	Blade of grass.	Abscess at the first rib.		Recovery.
Küpper, cited by Sander, *l. c.*	Man of thirty-two years.	Head of grain in left lung.	Pneumonia, then improvement, with stinging pains internally and offensive expectoration. A year later brain symptoms developed.	No treatment.	Died after one year.

SOFT, ROUGH BODIES. (HEADS OF GRAIN.)—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Mayo, cited by Sander, p. 343.		Head of grain.	Abscess of right lung, extending into liver.	No treatment.	Death.
Marrow, "Lancet," 1862.		Spear of hay.	After twelve weeks, symptoms of abscess. The abscess ruptured between the tenth and twelfth ribs.	No treatment.	Recovery.
Donaldson, S. J. B. 7, p. 177.	A young lady.	Head of grass in trachea or bronchi.	Attacks of coughing resembling pertussis with putrid expectoration. Object coughed up on fiftieth day.	No treatment.	Recovery.
Mandowski, "Deutsche med. Wochenschr.," 1895, No. 30.	Boy of eight years.	Head of grain in left bronchus.	Pleuropneumonia on left side. Object was coughed up.	No treatment.	Recovery.
Mandowski, "Deutsche med. Wochenschr.," 1895, No. 30.	Boy of one year.	Twig of Thuya.	A month later there was fever, with cough and expectoration at times in large quantities. (Opening of abscess.)		Gradually eight pieces of the sprig were coughed up. The child appears well.

BODIES THAT SWELL IN WATER. (FRUIT SEEDS.)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Barrand, "Jahrb. für Kinderkrankheiten," N. F., Bd. 24, p. 325.	Child of three years.	Bean in right bronchus.		Tracheotomy *in extremis*, on the day after the accident.	Death.

Bramwell, "Brit. Med. Jour.," 1879, vol. 25, p. 205.	Girl of seven years.	Bean in right bronchus.		Tracheotomy immediately, without success. Said to have been expelled in a fit of choking, three weeks later.	Recovery.
Bonderen, "Semon's Centralblatt," VIII, 1891.	Boy of three years.	Kernel of summer wheat in right bronchus.		Tracheotomy. Extraction with forceps.	Recovery.
Bonderen, "Semon's Centralblatt," VIII, 1891.	Girl of three and one-half years.	Kernel of corn, put in nose, fell into right bronchus.		No operation.	Death on twenty-second day.
Glesinger, 1877, S. J. B. 181, p. 60.	Boy of eight years.	Bean in right bronchus.		Emetics.	Recovery.
Klein, cited from Kühn, 1807.	Boy of ten years.	Bean in right bronchus.		Operation on same day, but unsuccessful.	Died after eight hours.
Sturm, cited from Kühn, 1857.		Bean in the upper of the three right bronchial branches.		Tracheotomy without success.	Died on the following day
Ulrich, cited from Kühn, S. J. B. 3, p. 177.	Child, three years old.	Sword bean in upper portion of left bronchus.		Unsuccessful tracheotomy.	Died during operation.
Albert, cited from Kühn.	Child of two years.	White bean in left bronchus.		No operation.	Died a short time later.
Bönten, cited from Kühn, 1834.	Child of four years.	Bean in left bronchus.		No operation.	Died on the ninety-first day.
Kopf, cited from Kühn, 1845.		Bean in the right lateral branch of left bronchus.		No operation.	Died after sixty days.
Möller, 1835, S. J. B. 15, p. 72.	Child of four and a half years.	Bean firmly fixed in left bronchus.		No operation.	Died after two days.

Bodies that Swell in Water. (Fruit Seeds.)—(*Continued.*)

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Rujer, cited from Kühn.		White bean in right bronchus.		No operation.	Recovery.
Schütz, S. J. B. 14, p. 210	Child of three years.	Piece of apple seed in right bronchus.		No operation.	Suffocated immediately.
Hamburger, "Berliner klin. Wochenschr.," 1873, 28 und 29.	Man of seventy years.	Green pea in right bronchus.	Dyspnea, loss of memory, loss of appetite, hebetude. It was coughed up on the eighth day, after using emetics.		Recovery.
Sanders, "Deutsches Archiv für klin. Med.," vol. 16, p. 330.	Woman.	White bean in right lung.	The presence of a foreign body was not recognized. Symptoms of a protracted pneumonia; after some months, bean was coughed up.		Recovery.
Porter, 1822, S. J. B. 75, p. 75.	Boy of nine years.	Turkish bean in right bronchus.		Tracheotomy at first unsuccessful. Then the object was dislodged by the introduction of a long sound.	Recovery.
Hager, M. 51.	Girl, seven years old.	Bean in left lung.		Tracheotomy at first unsuccessful. Then the body was dislodged by the introduction of a blunt hook.	Recovery.
Gross, cited by Sander, p. 343.	Man of forty-eight years.	Hawberry in right bronchus.	Hectic fever, cavity formation.	No operation.	Death after thirteen months.
Bertholle, ibid., p 347.	Girl of six years.	Bean in left bronchus.		Easily movable. Tracheotomy after six days. Bean was coughed up.	Recovery.
Wagner, "Arch. für Heilkunde," v, p. 347.	Boy of four years.	Bean in left bronchus.		Easily movable. Fits of choking with cyanosis.	Died after two days in a fit of choking.

Ibid.	Girl of three years.	Bean.	Bronchitis developed.	Tracheotomy.	Died after seven days.
Armstrong, cited by Sander.	Boy, ten years old.	Bean.		Tracheotomy. Extraction with forceps.	Recovery.
Bourdillat, cited by Sander.	Child, seven years old.	Bean in right lung.	A peculiar fluttering can be heard and felt over the larynx.	Tracheotomy and introduction of a dilator.	Recovery.
Cruveilhier, from Poulalion, p. 178.	Woman.	Kernel of maize.	Violent attacks of coughing and choking. After five months, it became calcified and was coughed up.		Recovery.
Leoni and Hoch, S. J. B. 93, 1857, p. 337.	Boy of nine years.	Bean.	Had a fit of choking and then felt well. After six days an extremely severe attack occurred. Right side of chest less movable than left. On the fifteenth day another attack and then there was no respiration on the left. Bean was coughed up.		Recovery.
Grun, "Lancet," 1889, II, p. 1224.	Child of two years.	Pea in left bronchus.	Inflammation of left lung.	No operation.	Death four weeks later.
Moure, S. J. B. 225, p. 160.	Girl, seven years, old.	Kernel of maize in right bronchus.		Bronchopneumonia. No operation.	Death.
Goodhart, "Brit. Med. Jour.," March, 1886, p. 590.	Child.	Bean.	A pleuritis was diagnosed and the child died with symptoms of meningitis. An encapsulated empyema was found and a group of much enlarged bronchi; the bean was found occluding one of the bronchi.		Death.

HARD FRUIT SEEDS THAT DO NOT SWELL IN WATER.

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Spince, cited from Kühn, 1859.	Boy, four years old.	Plum-stone in left bronchus.		Unsuccessful tracheotomy.	Death on the following day

HARD FRUIT SEEDS THAT DO NOT SWELL IN WATER.—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Ziis, cited from Kühn, 1858.	Boy.	A plum-stone cut into a whistle.	No symptoms.		Recovery (?).
Spince, cited from Kühn, 1859.	Girl.	Plum-stone in left bronchus.		Unsuccessful tracheotomy immediately.	Death a few days later.
Kobler, "Wiener klin. Rundschau," 1895.	Woman of forty-five years.	Plum-stone.	Pleuritis developed. The stone was coughed up.		Recovery.
Hiss, Dissertation, Greifswald, 1891.	Child.	Cherry-pit in right bronchus.	Bronchopneumonia developed. Later the pit became loosened; severe paroxysm of coughing, dyspnea, convulsions.	No operation.	Death.
Eggerdes, from Poulalion, Thèse de Paris, 1890–91, p. 178.	Man.	Cherry-pit.	Nothing was known of the presence of the foreign body. Phthisis developed. The pit became calcified and was coughed up with much pus.		Death.
Maslieurat Lagumard, ibid.	Girl.	Cherry-pit.	Violent attacks of coughing and choking. The pit became calcified and was coughed up eight months later.		Recovery.
Masen, from Poulalion, p. 179.	Man of forty-five years.	Cherry-pit.	Phthisis. The pit became calcified and was coughed up some days before death.		Death.
Mackay, "Brit. Med. Jour.," March 23, 1889, p. 657.	Boy of three years.	Pieces of a plum-stone in left bronchus.	Chronic pneumonia.	No operation. Emetics were without effect.	Death.
Leoni and Hoch, S. J. B. 93, 1857, p. 337.	Boy of four years.	Cherry-pit.	A painful sensation on one side of chest. Nineteen years later, fever, pain, and the pit was coughed up with much pus.		Recovery.
Simon, "Brit. Med. Jour.," Nov., 1887, p. 998.	Boy of six years.	Plum-stone in right bronchus.		Tracheotomy.	Recovery.

Easles, "Brit. Med. Jour.," Oct., 1885, p. 669.	Boy of two years eight months.	Cherry-pit in left bronchus, 3 cm. behind bifurcation.		Emetics. Inversion; tracheotomy thirty-five hours after the first attack; unsuccessful.	Death half-hour after operation.
Harrison, "Brit. Med. Jour.," June, 1888, p. 1337.	Girl of eight years.	Plum-stone in right bronchus.	Inspiratory and expiratory râle heard best at the fourth dorsal vertebra.	Tracheotomy on seventeenth day. Stone was coughed up immediately.	Recovery.
Mandowski, "Deutsche med. Wochenschr.," 1895, No. 1.	Man of thirty years.	Piece of almond in left bronchus.		It was coughed up on the patient's assuming position according to directions.	Recovery.
Steinfinkel, "Arch. für Kinderheilkunde," III, 1882.	Child of two years.	Pieces of almond in both bronchi.	Emetics. The child was held by the feet and swung to and fro.		Death on second day.

OTHER FOREIGN BODIES.

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Prati, "Sperimenti," November, 1879.	Man.	A living fish in right bronchus.			Immediate death.
Maschka, "Prager med. Wochenschr.," 1890, p. 46.	Man.	Masticated meat, in trachea and left bronchus.			Immediate death.
"Froriep's Notizen," vol. 49, p. 254.	Girl of nine years.	Thread-worm in trachea and right bronchus.	Choking.	No operation.	Death on thirty-first day.
Blandin, cited from Kühn.	Child.	Thread-worm in right bronchus.	Choking.	No operation.	Death.
Rocher et Tardieu, 1859, S. J. 104, p. 373.		A morsel of food in right bronchus.	Paralysis (diphtheria) of the pharynx developed.	No operation.	Immediate death.

OTHER FOREIGN BODIES.—(*Continued.*)

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Ryland, S. J., vol. 19, p. 143.	Adult.	Pieces of food in nearly all parts of air-passages.		No operation.	Immediate suffocation.
Ast, "Münchener med. Wochenschr.," 1892, p. 605.	Child of four years.	Piece of spice in right bronchus.	Pneumothorax of right side. Object was coughed up.		Recovery.
Bryant, "Brit. Med Jour.," June, 1889, p. 1348.	Man forty-nine years old.	Button aspirated during sleep.	Attack of dyspnea, which quickly passed away; chronic thoracic symptoms.	Operation refused.	Death after about eight weeks.
Kobler, "Wiener klin. Wochenschr.," 1889, No. 33.		Shirt button.	No pain at first.		Death.
Kobler, "Wiener klin. Rundschau," 1895.	Child of two and a half years old.	Essence of vinegar passed into air-passages. Corrosion of left bronchus much greater than of right.			Death sixteen hours later.
Langerhans, "Deutsche med. Wochenschr.," 1893, No. 12.		Carbolic acid, in a case of poisoning. Corrosion much greater in right bronchus.			Death.
Sander, "Deutsches Arch. für klin. Medicin," vol. 16, p. 330.	Man of thirty-nine years.	Shirt button.	Sensation as of body passing down into right side. Fever with purulent expectoration. Object coughed up after six and a half months.	No treatment.	
Da Costa, "Virchow-Hirsch's Jahresb.," 1888, II, p. 229.		Piece of apple.	Bronchial asthma. Object was coughed up.		Recovery.

Pitts, "Lancet."	Boy of nine years.	Piece of chestnut in right bronchus.	A piece was extracted from larynx. Pneumonia of right upper lobe and cavity formation.		Partial recovery.
Ruhsmore, "Semon's Centralblatt," VIII.	Man.	Cork of medicine bottle in left bronchus.		Tracheotomy.	Death.
Broadbent, "Brit. Med. Jour.," Sept., 1880, p. 509.		A piece of maple twig, branched.	Formation of various metastatic abscesses.		Death.
Bryant, *l. c.*	Boy of seven years.	Mouthpiece of trumpet in left bronchus.		On the thirteenth day, tracheotomy. Seized with forceps which had to be passed down 14 cm.	Recovery.

[Herewith are added the cases which have been recorded since the publication of the original text. Ninety-two have been abstracted from the original reports. An analysis follows.]

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Wade, "Lancet," London, 1895, II, 1499.	Boy, five years.	Tamarind stone in right bronchus.	Coughing. Inspiratory stridor. No air going to right lung. Fever.	Tracheotomy.	Death.
Morgan, "Lancet," London, 1895, II, 769.	Girl, eight years.	Part of plum-stone in left bronchus.	Pain in neck. Cough. Dulness of left chest.	Tracheotomy.	Recovery.
Winands, "München. med. Wochen.," 1895, XIII, 1211.	Child, three years.	Bean in right bronchus.	Cough. Dyspnea. Unconsciousness. Pain in right chest.	Emetic. Patient held upside down.	Death.
Mackenzie, "St. Thomas's Hosp. Rep.," London, 1892–93, N. S., XXII, p. 137.	Boy, three years ten months.	Clove in right bronchus.	Vomiting. Fever. Anorexia. Bronchopneumonia. Abscess of lung.	None.	Death.

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Crommelin, "Australas. Med. Gaz.," Sydney, 1894, XIII, p. 336.	Girl, three years.	Grain of corn in right bronchus.	Coughing. Dyspnea. Diminished breath sounds over right chest.	Tracheotomy.	Recovery.
Murray, "Ann. Surg.," Phila., 1895, XXII, p. 519.	Boy, seven years.	Metallic end of lead-pencil. Left bronchus.	Choking. Dyspnea. Absence of respiratory murmur in upper left lung.	Coughed up.	Recovery.
Homans, "Boston M. & S. Jour.," 1895, CXXXIII, p. 164.	Boy, three years.	Brass safety-pin. Right primary bronchus.	Dyspnea.	Laryngotomy.	Recovery.
Koch, "Ann. de Mal. de l'Oreille et du larynx," Paris, 1896, XXII, p. 11, 295–307.	Boy, nine years.	Bean in left bronchus.	Dyspnea. Fever. Dulness of left lung.	Tracheotomy.	Recovery.
Thomas, "Rev. Méd. de la Suisse Rom.," Genoa, 1896, XVI, p. 246.	Boy, eleven months.	Piece of leek. Left bronchus.	Cough. Dyspnea.	Tracheotomy.	Recovery.
Archer, "Lancet" London, 1897, I, 1146.	Boy, nine years.	Piece of nut. Bronchi.	Fever. Cough. Pain in left chest. Abscess.	No operation.	Recovery. Coughed up.
Marshall, "Brit. Med. Jour.," London, 1897, I, p. 1158.	Girl, four and a half years.	Piece of slate pencil ¾ inch long in left bronchus.	Vomiting. Hemoptysis. Dyspnea. Cough. Fever.	No operation.	Recovery. Coughed up after twenty-seven days.
Francis, "Brit. Med. Jour.," London, 1897, II, 809.	Man.	Pipe-stem 1½ inches long in left bronchus.	Dyspnea. Pain in left breast. Cough.	No operation.	Recovery. Coughed up after three months.

Godlee, "Med. Chir. Tr.," London, 1896, lxxix, 197–207.	Boy, seventeen years.	Part of ear of corn. Left bronchus.	Hemoptysis. Empyema.	Thoracoplasty.	Death due to abscess of brain.
Himmelsbach, "Buffalo Med. Jour.," 1897–98, xxxvii, p. 19.	Woman, twenty-one years.	Tooth in left bronchus.	Dyspnea. Coughing. Bronchitis. Hemoptysis.	No operation.	Recovery. Tooth coughed up four months after.
Hough, "Quart. Med. Jour.," Sheffield, 1896–97, v, 245–248.	Man, thirty-five years.	Pipe-stem 2 inches long in left bronchus.	Cough. Hemoptysis. Sibilant rhonchi over left upper lung.	None. Coughed up.	Recovery. Death due to general paralysis.
Crumbine, "Cincin. Lancet and Clinic," N. S., xxxvii, 648.	Boy, two years seven months.	Nail 2 inches long. Right lung.	Vomiting. Lobar pneumonia. Coughing. Abscess of lung.	Tracheotomy.	Recovery. Coughed up after nine months.
"Yale Med. Jour.," New Haven, 1896–97, iii, 137.	Boy, six years.	Grain of corn. Right bronchus.	Dyspnea. Caught in a fit of coughing. Kernel of corn dislodged and into esophagus and passed per rectum four hours later.		Recovery.
Kollofrath, "München. med. Wochen.," 1897, xliv, 1038.	Man, sixty-three years.	Piece of bone. Right bronchus.	Dyspnea. Cough. Nausea.	Removed with forceps after tracheoscopy.	Recovery.
Godlee, "Med. Chir. Tr.," London, 1896, lxxix, 197–207.	Boy, sixteen years.	Peg of a peg top. Air-passage of left side.	Empyema. Seven and a half years after swallowing peg. Bronchiectasis. Hemoptysis.	Tracheotomy. Opening of pleura. One and a half years after peg came out of wound.	Recovery.
Ibid.	Boy, six and a half years.	Small ivory knob. Left bronchus.	Bronchiectasis. Dulness in left side.	No operation.	
Ibid.	Boy, four years.	Portion of vertebra of a rabbit.	Bronchiectasis. Coughed up the body ten months after operation.	Thoracoplasty.	Improvement.
Fulton, "Med. Rec.," N. Y., 1897, li, 264.	Boy, seven years.	Nail 1½ inches long. Air-passages.	Coughing.	No operation. Coughed up.	Recovery.

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Binswanger, "Med. Sentinel," Portland, Ore., 1896, IV, 329.	Man.	Dime. Right bronchus.	Coughing.	Coughed up after three months.	Recovery.
Fronz, "Jahrb. f. Kinderheil.," Leipzig, 1898, XLVII, 74–89.	Child, three years.	Screw. Bronchus.	Empyema.	Thoracoplasty. Screw came out one and a half years after it was swallowed.	
Schiffers, "Ann. Soc. Med. Chir. de Liége," 1897, XXXVI, 75.	Boy, three years.	Portion of vertebral column of fish in larynx.	Suffocation.	Tracheotomy.	Recovery.
Java, "Med. Record," Oct. 7, 1899, p. 514.	Man, seventeen years.	Tack $\frac{7}{8}$ inch long in lung.	Cough for seven years. Hemoptysis.	No operation.	Recovery.
Bonnus, "Bull. et. Mém. Soc. Méd. des. Hôp. de Paris," 1897, 3 s., vol. XIV, 1214–1217.	Girl, five years.	Piece of a black glass bead 1 cm. long in trachea.	Dyspnea. Cough. Bloody sputum.	Intubation.	Recovery. Bead coughed up through tube.
Mathew, "Brit. Med. Jour.," 1901, vol. I, p. 888.	Girl, five years.	Tack in lung, $\frac{1}{3}$ inch long.	Empyema. Lobar pneumonia.	Thoracoplasty.	Recovery. Tack coughed up two years after being swallowed.
Moscucci, "Riforma Med.," Naples, 1898, XIV, 124–127.	Man, thirty-six years.	Ascaris lumbricoides in trachea.	Vomiting. Dyspnea. Convulsions.	No operation.	Death.

Warrack, "Brit. Med. Jour.," 1899, vol. I, p. 401.	Woman, twenty-six years.	Tooth in left bronchus.	Dyspnea. Cough. Cyanosis. Immobility of left chest. Gangrene of left lung.	No operation.	Death.
Vertogradoff, "Voyenno Med. Jour.," St. Petersb., 1898, CXCII, 651–662.		Pumpkin-seed in left bronchus.	Bronchopneumonia of left side.	Laryngotracheotomy.	Recovery.
Peyrissac, "Rev. Hebdom. de laryngol.," etc., Paris, 1898, XVIII, 1–14.	Man, eighteen years old.	Prune-pit in left bronchus.	Dyspnea. Cough.	Injection of cold water through larynx into trachea.	Recovery. Stone coughed up.
Nordman, "J. de Méd. et Chir. prat.," Paris, 1897, LXVIII, 772–775.	Girl, eight years.	Skin balloon 2½ cm. long in right bronchus.	Cough. Dyspnea.	Tracheotomy.	Recovery.
Meyjes, "J. Laryngol.," Lond., 1897, XII, 599–602.	Woman, twenty-one years.	Needle 3 cm. long in larynx.	Pain in larynx.	Removed by forceps.	Recovery.
Quincke, "Mitteil. aus den Grenzgeb. der Med. und Chir.," 1895, vol. I, 47, 54, 55.	Man, forty-seven years.	Chicken-bone in right lung.	Bronchiectasis. Abscess.	Thoracoplasty.	Recovery. Bone coughed up.
Félizet, "Bull. et Mém Soc. de Chir. de Par.," 1898, N. S., XXIV, p. 524.	Child, five years.	Part of slate-pencil in trachea.	Coughing.	Tracheotomy.	Recovery.
Hue, ibid., p. 1021.	Child.	Whistle 3 cm. long. Left bronchus.	Suffocation. Vomiting.	Tracheotomy.	Recovery.
Horn, "Jour. Am. Med. Ass.," Chicago, 1898, XXXI, 137.	Boy, twelve years.	Pin. Left bronchus.	Cough. Dyspnea. No breath-sound over left lung.	Tracheotomy.	Recovery.

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Bunch and Lake, "Lancet," London, 1897, ii, 784–786.	Woman, thirty-eight years.	Small piece of bone in right bronchus and trachea for nine years.	Cyanosis. Dyspnea. Cough. Hemoptysis. Bronchitis.	Tracheotomy.	Recovery.
Thompson, "Nat. Med. Rev.," Wash., 1896–97, vi, 280, 281.	Child, three years.	Watermelon seed in trachea.	Dyspnea. Cough.	Tracheotomy.	Recovery.
Ibid.	Boy, fifteen months.	Small stone. Trachea.	Cough. Cyanosis.	Tracheotomy.	Recovery.
Luch, ibid.	Woman, forty-five years.	Chicken - bone. Left bronchus.	Coughing.	No operation. Coughed up after four months.	Recovery.
Haussay, "Arch. de Méd. d. enf.," Paris, 1899, ii, 99–101.	Child, thirty months.	Ascaris lumbricoides 20 cm. long. Trachea. Also a part of prune-pit in larynx.	Dyspnea. Cyanosis.	No operation.	Death.
Félizet, "Bull. et. Mém. Soc. de Chir. de Paris," 1898, N. S., xxiv, 522.	Girl, nine and a half years.	Prune-stone. Left bronchus.	Suffocation. Lobar pneumonia.	Tracheotomy.	Death.
Ibid., p. 524.	Child, eight years.	Button. Air-passages.	Suffocation.	No operation.	Death.
Ibid.	Child, four years.	Small key in trachea.	Suffocation.	Tracheotomy.	Recovery.
Marmasse, "Rev. prat. d'Obst. et de Pædiat.," Paris, 1897, x, 80–82.	Boy, two years.	Pebble 1 cm. long. Trachea	Suffocation. Bronchopneumonia.	Tracheotomy after intubation had been tried.	Death.

Ponchon, ibid., 1897, x, 25–30.	Girl, nine years.	Part of a nut. Larynx.	Suffocation.	Tracheotomy.	Recovery.
Noltenius, "Arch. f. Laryngol. a. Rhinol.," Berlin, 1897, vi, 154–156.	Boy, four years.	Glass bead. Left bronchus and trachea.	Characteristic murmur in trachea. "Flopp" suffocation.	Tracheotomy.	Recovery.
Battle, "North Car. M. J.," Winston, 1898, xli, 409–413.	Girl, sixteen years.	Shawl pin 1¾ ins. long. Trachea and larynx.	Cough. Bloody sputum.	Removed with forceps after cutting in half twenty-two days after swallowing.	Recovery.
Wyman, "Physician and Surg.," Detroit and Ann Arbor, 1897, xix, 198–207.	Man.	Ten-cent piece. Air-passage.	Dyspnea. Cyanosis	Patient suspended by feet.	Recovery. Dime fell out of mouth
Massei, "Arch. ital. di laringol.," Naples, 1896, xvi, 125–133.	Boy, nine years.	Pumpkin-seed in trachea.	Suffocation.	Removed with forceps.	Recovery.
Heindl, "Wien. klin. Wochenschrift," 1896, ix, 832.	Man, twenty-three years.	Needle 3.5 cm. long in larynx.	Pain in the throat.	Removed with forceps.	Recovery.
Koch, "Wiener klin. Rundschau," 1897, ix, 72–74.	Boy, nine years.	Bean in larynx and left bronchus.	Suffocation. Atelectasis of left lung.	Tracheotomy.	Recovery.
Ibid.	Boy, six years.	Piece of bone in larynx.	Aphonia. Pallor. Acute stenosis of larynx.	Tracheotomy.	Recovery.
Ibid.	Woman, forty years.	Piece of bone in trachea.	Suffocation.	Laryngotracheotomy eighty days after.	Recovery.
Ibid.	Woman, twenty-seven years.	Pin in larynx.	Suffocation. Pain.	Tracheotomy.	Recovery.
Ibid.	Child, eight years.	Small bean in left bronchus.	Suffocation.	Tracheotomy.	Death from pleuropneumonia.

Author, Reference.	Patient.	Foreign Body (Location).	Symptoms.	Treatment.	Result.
Briquet, "Jour. de Méd. et Chir. Prat,," Paris, 1898, LXIX, 369–372.	Child, three years.	Button. Air-passages.	Cough. Bronchitis.	No operation.	Death a year after swallowing.
Lavrand, "Jour. d. Sc. Méd. de Lille," 1897, I, 32–35.	Boy, ten years.	Rosary bead in the right bronchus and trachea.	Cyanosis. Cough. Dyspnea.	Tracheotomy.	Recovery.
Heller, "München. med. Wochenschr.," 1898, XLV, 837.	Girl, ten years.	Part of turnip in trachea.	Suffocation.	No operation. Coughed up after douche of nasopharynx.	Recovery.
Ibid.	Child, one year.	Almond in right bronchus.	Pneumonia.	No operation. Coughed up after douche of nasopharynx.	Recovery.
Ibid.	Man, sixty-five years.	Prune-stone. Air-passages.	Orthopnea. Cyanosis.	Coughed up after douche of nasopharynx.	Recovery.
Buberl, "Wien. med. Wochenschrift," 1896, XLVI, 1524–1528.	Man, thirty-eight years.	Piece of wood in right bronchus.	Cough. Hemoptysis. Fever. Dulness of right chest.	No operation.	Death by perforation of body into right branch of pulmonary artery.
Aronsohn, "Berlin. klin. Wochenschr.," 1895, XXXII, 989, 1899.	Man, forty years.	Piece of bone in left bronchus.	Bronchitis.	No operation.	Recovery after body was coughed up.
Ibid.	Woman, thirty-five years.	Limestone—*i. e.*, lung-stone—in trachea, entered from within.	Cough. Mucopurulent expectoration, at times bloody.		Recovery.

Hall, "Univ. Med Mag.," Phila., 1895-96, VIII, 527	Girl, eleven years.	Small stone in right bronchus.	Coughing. Bronchitis.	Stone coughed up five days after.	Recovery.
London, "Intercolon. M. J. Australas.," Melbourne, 1898, III, 132-142.	Boy, seven years.	Shirt-stud in right bronchus.	Suffocation. Pain in chest. Wheezing. Sonorous rhonchi over right lung. Hemoptysis. Pneumonia. Hectic fever. Abscess of lung.	Thoracoplasty. Lung was punctured and abscess drained.	Recovery. Stud coughed up after operation.
Packard, "Tr. Path. Soc. Phila.," 1898, XVIII, 273-275.	Man, forty-two years.	Fragments of coke in right bronchus and in left bronchus.	Cyanosis. Fever. Dyspnea. Moist râles and tubular breathing over both lungs in spots. Bronchopneumonia.		Death from cardiac and respiratory failure.
Havemann, "Deutsche Zeitschr. f. Chir.," Leipzig, 1898-99, L, 401-403.	Girl, five years.	Bean in right bronchus.	Cough. Pain in neck. Suffocation. Dulness of part of right side. Fever. Dyspnea.	Tracheotomy. Bean gotten out with sharp spoon.	Recovery.
Mandowski, "Deutsche med. Wochenschr.," 1895, XXI, 479-481.	Girl, three years.	Needle 6 cm. long. Left bronchus.	Cough. Pain in left side of neck. Blood coughed up. Dulness and bronchial breathing left lung. Fever. Pneumonia.	No operation.	Death from pneumonia four months after.
Ibid.	Boy, seven years.	Small twigs in air-passages.	Cough. Fever. Hemoptysis, some dulness of left lung.	No operation.	Recovery. Twigs coughed up gradually.
Ibid.	Boy, eight years.	Grain of barley and twig 5 cm. long in air-passages.	Suffocation. Cough. Fever. Pain in left side. Bloody purulent sputum. Pleuropneumonia.	No operation.	Recovery after coughing up body.
Ibid.	Man, thirty years.	Almond in air-passages.	Cough. Dulness over part of left lung.	No operation.	Recovery after body was coughed up.
Ibid.	Man, sixty-four years.	Piece of mutton in larynx and trachea.	Suffocation.	No operation.	Death.

AUTHOR, REFERENCE.	PATIENT.	FOREIGN BODY (LOCATION).	SYMPTOMS.	TREATMENT.	RESULT.
Glasgow, "Virginia Med. Monthly," Richmond, 1895–96, XXII, 55–58.	Girl, sixteen years.	Hat pin 2¼ inches long in trachea.	Dyspnea. Coughing.	Tracheotomy.	Recovery.
Carslaw, "Glasgow M. J.," 1895, XLIII, 274–280.	Boy, eight years.	Plum-stone in air-passages.	Coughing. Pneumothorax.	Tracheotomy.	Recovery. Stone coughed out five weeks after aspiration.
Curtis, "Ann. Surg.," Phila., 1898, XXVIII, 605–610.	Boy, eleven years.	Pin through a berry in right bronchus.	Coughing. Pneumonia.	Tracheotomy failing, then posterior thoracotomy.	Death from pneumonia.
Rogers, "Memphis M. Month," 1895, XV, 97–102.	Boy, eight years.	Cockleburr.	Coughing.	Laryngotracheotomy.	Recovery.
Ibid.	Child, thirteen months.	Grain of corn in trachea.	Coughing. Râles. Fever.	Laryngotracheotomy.	Recovery.
Verco, "Australas. M. Gaz.," Sydney, 1898, XVII, 209–211.	Girl, three years.	Watermelon-seed in trachea.	Cough.	Tracheotomy.	Recovery.
Ibid.	Boy, three years.	Watermelon-seed in larynx.	Laryngeal stridor. "Flapping" sound in larynx, convulsions. Impaired resonance at right base.	Seed coughed up after five months.	Recovery.
Ibid.	Woman, twenty-seven years.	Tooth in small bronchus of right side.	Cough. Fever. Impaired resonance over right lung posteriorly. Purulent sputum.	Tooth coughed up after twelve weeks.	Recovery.
Ibid.	Woman, thirty-nine years.	Tooth in bronchus.	Consolidation of upper right lung, followed by ulceration of bronchus, and abscess after tooth coughed up.	Tooth coughed up.	Death.

Landau, "Therap. Wochenschr.," Wien, 1896, III.	Man, thirty-six years.	Nutshell in larynx.	Cough. Hemoptysis. Dyspnea.	Coughed up one year and three months after.	Recovery.
Gomez de la Mata, "Ann. d. Mal. de l'oreille du larynx," Paris, 1898, XXIV, 636-640.	Child, four years.	Bean in larynx and then in right bronchus.	Dyspnea. Edema of lungs.	None.	Death.
Wigg, "Australas. M. Gaz.," Sydney, 1898, XVII, 171.	Boy, four years.	Piece of sheep's rib in larynx.	Dyspnea. Cyanosis. Shock.	Tracheotomy.	Recovery.
Ibid., p. 172.	Woman, thirty years.	Tooth in small bronchus on right side.	Coughing. Soreness at lower end of sternum. Blood and pus coughed up. Slight dulness of base of right lung. Empyema. Abscess of lung.	Tracheotomy.	Death.
Swift, ibid., p. 172.	Child, two years.	Screw in right bronchus, and then in trachea.	Coughing. Dyspnea. Impaired movement of right chest.	Tracheotomy.	Death.
Ibid.	Child, three and a half years.	Piece of nutshell in trachea.	Dyspnea. Cough.	Tracheotomy.	Recovery.
Gibson, "Australas. M. Gaz.," Sydney, 1895, XIV, 47-50.	Boy, five years.	Grain of corn in trachea.	Cough. Choking. Dyspnea. Thirst. Anorexia. Stridulous breathing. Orthopnea. Resonance diminished right side anteriorly. Ronchi.	Tracheotomy.	Recovery.
Ibid.	Girl, three years.	Grain of corn in trachea.	Cough. Choking. Impaired resonance in left side. Breath sounds deficient over lower half of left lung.	Tracheotomy.	Recovery.

[This addition of cases modifies slightly the conclusions of the author as to results of treatment, as the analysis at the end of the article indicates.—Ed.]

It is in the right bronchus that foreign bodies are most frequently found, because its course is much more nearly vertical than that of the left, and it therefore affords a better opportunity for the entrance of a foreign body. This is readily understood when the larger size of the right lung and right bronchus, and consequent greater power of aspiration, are taken into account. Kobler rightly calls attention to the fact that the position of the individual must affect the movement of the foreign body. If the patient's body is inclined to the right, the entrance into the left bronchus is necessarily facilitated.

It may also happen that a foreign body enters one bronchus and, after being thrown back into the trachea, finds its way into the other bronchus; a case occurs now and then in the literature which can be explained in no other way.

SYMPTOMS.

The presence of a foreign body in the bronchi may cause a great variety of symptoms, according as the body is fixed or movable, as the occlusion of the bronchus is complete or only partial, and as the patient's sensibility is preserved or abolished. In many cases, after an initial violent fit of coughing, weeks and months may elapse during which the patient's comfort is not disturbed. The symptoms of the initial period will be enumerated first:

1. **Diminution or Complete Abolition of the Respiratory Sounds on One Side of the Chest.**—If the bronchus is completely occluded, the absence of respiration may be so marked that it practically suffices to establish the diagnosis. If, however, the bronchus is only partially occluded, the difference between the two sides may be very slight, or even quite unrecognizable. It is evident, therefore, that a foreign body which fits into the bronchus may cause more marked symptoms than a hard angular body which allows some air to pass.

2. **Diminution or abolition of respiratory movement** on the affected side is connected with, and immediately dependent upon, the foregoing symptom. This sign in itself is, of course, quite as important as the preceding; but as consolidation soon develops in many cases, it is often rendered worthless, or at least of very doubtful value for the diagnosis. Signs of pneumonia may make their appearance as early as twenty-four hours after the entrance of the foreign body. In Finkelnburg's case no sonorous râles could be heard over the area of dulness, which was quite marked; he considered the phenomenon due to the accumulation of secretions in a space cut off from communication with the air. The *vocal fremitus* is also increased over such an area of consolidation.

It is to be noted that *consolidation* is not a necessary consequence of the entrance of a foreign body. In a number of cases, at least during the first days, atelectasis only develops in the affected portion of the lungs. The percussion sound may be somewhat dull, although dulness may also be absent or at least very insignificant. This obviously depends on the amount of air remaining in the lung, which, again, bears a fixed relation to the manner in which the foreign body occludes the bronchus. It is impossible at the present time to discuss the subject in all its details, as these finer distinctions are not recorded in the case-histories.

3. **Sibilant or sonorous rales** heard over the suspected site of the foreign body are especially important, if they are heard on one side, in the interscapular space. The râles may be very loud, and a *thrill*, which may be transmitted to the trachea,* is sometimes felt if the hand is applied to the area.

Sometimes the nature of the foreign body greatly favors the production of a sound; for instance, the whistling sound heard in the case in which a whistle had been aspirated.

4. **Pain and Paresthesia.**—Pain is complained of only when the body remains in the same place for some time and sets up an inflammatory process. As a rule, however, the patients at once complain of a feeling of pressure and uneasiness; they often say they feel as if the object were moving.

There are also cases in which severe and characteristic pain immediately follows the entrance of the foreign body if its size is such as to cause violent distention of the tissues. The patient's statements on this point must, however, be accepted with great caution; there are cases in which the pain was referred to the chest when the foreign body was in the larynx, and vice versa.

5. **Dyspnea.**—Dyspnea sets in at once, but the respiration is not at all, or only slightly, accelerated. There is some interference with both inspiration and expiration; one of the two is usually very labored, but it may be either one or the other. Judging from what occurs in analogous cases in the trachea, it might be supposed that inspiration would be chiefly affected; but it is expressly stated in some cases that expiration was chiefly affected, and this is also true of some cases of tracheal stenosis.

Although the dyspnea is, as a rule, continuous, there are also a number of cases characterized by occasional exacerbations, and Da Costa, in speaking of his case, in which a piece of apple had entered the air-passages, directly mentions bronchial asthma. The foreign body was evidently movable; it is also quite possible that the dyspneic attacks excited by local irritation at various points of the respiratory tract may closely resemble bronchial asthma. In a large number of cases ominous attacks of asphyxia were reported.

These attacks of suffocation are often very severe and directly threaten the patient's life; they are particularly apt to occur during the initial period, before the foreign body has become firmly fixed, so that it can be driven against the glottis or gives rise to irritation at the region of the spur in the bifurcation. Gross has given a vivid description of such a case. The patient is seized with a feeling of approaching dissolution, he gasps for air, looks wildly about him, and perhaps loses consciousness. The face is livid, the eyes are prominent, the heart beats tumultuously, and the body twists and turns in all directions. Foam, often mixed with blood, exudes from the nose and mouth; at times he grasps at his throat, utters piercing screams, or falls into a condition of unconsciousness. Sometimes vomiting follows, and with it there may be an amelioration of the symptoms. Involuntary evacuation of urine and feces may occur.

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* In one case the râles, which could be heard over the sound side, were endowed with a peculiar timbre by the presence of the thrill; on the sound side the phenomenon was entirely absent, as evidently no air could penetrate, and the vibrations were therefore not propagated.

6. The **sputum** is usually purely catarrhal, but blood may be mixed with it in small quantities. Finally, there may be cases in which there is an abundant expectoration (expiration) of blood. The foreign body in such a case is sharp, or it has remained in the same place a long time and caused erosion of a large vessel. Hemorrhage was the cause of death in some cases.

7. **Loss of consciousness** is observed after sudden occlusion of a large bronchus.

8. **Nausea.**—Sanders describes in his own case a peculiar feeling of nausea, as in seasickness, after taking only a small quantity of nourishment.

9. **Peculiarities of Position and Attitude on the Part of the Patient.**—Few observations on this point have been recorded; in many cases the attitude is not significant and not at all influenced by the presence of the foreign body. But there are cases in which the patients are easy only when lying on their backs, as in Schrötter's case,* in which the breathing became laborious as soon as the patient sat up. In this case the foreign body (a needle) was evidently very movable, and this is a factor of very considerable importance as regards the attitude and actions of the patient. He will, so far as possible, assume a position in which the foreign body remains quiet.

Finally, it must be pointed out that all these symptoms may be absent. It is always strange how differently different individuals react to this injury. It is the general rule that a fit of coughing and choking occurs at the moment of entrance of the foreign body. However, objects of considerable size have entered without exciting a suspicion of their presence. If the foreign body remains movable, these convulsive attacks are repeated from time to time; but often they become more and more rare, although the body remains movable.

The attacks may also persist when the foreign body is not movable; or they may entirely disappear and both patient and physician may believe that the body is not in the air-passages, but has passed into the esophagus.

But even if all symptoms are lacking, disastrous consequences will gradually develop, and the presence of a foreign body must be inferred from these alone. [Paroxysmal cough is noted in most of the cases, and is more common than the author indicates.—Ed.]

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* *Wiener med. Jahrbuch*, 1868, p. 61.

The ultimate consequences of the presence of a foreign body in a bronchus depend much less on its size and form than on certain attendant circumstances. According to our present knowledge, we must ask: Was the body, which forced its way in, aseptic, or practically aseptic, or was it capable of causing infection?

Schüller * has administered to rabbits through tracheal wounds different foods, some clean and some mixed with filth, blood, and bacteria. While the pure foods were practically harmless, fatal pneumonia quickly followed the administration of the impure foods. We find that fine pieces of food are absorbed even in the bronchi and alveoli, and, if they are harmless in themselves, are tolerated by the body. Absorbable materials cause only a simple catarrh; those which cannot be removed lead to chronic cirrhotic inflammation, if they do not produce an infection.

According to these experiments, we can divide the consequences observed in man into *simple catarrhal, chronic inflammatory,* and *infectious.*

The first are frequent enough and may pass away completely after some weeks, when the foreign body is finally disposed of. In general, however, it is the rule that a chronic inflammation results, if the foreign body remains more than two or three days.†

In addition to bronchitis, masses of growing connective tissue develop in the deeper layers of the bronchus and in the adjacent lung tissue; the final result is the formation of a circumscribed cirrhosis about the foreign body as a central point. This process may be very slight, or it may, under certain circumstances, attain enormous dimensions, and a whole lung has been found changed into a cavernous meshwork. The relations may be so changed that even at the autopsy no clear idea can be gained.

Thus, Kobler relates the case of a man who was quite suddenly taken ill with cough and increasing cachexia. No dulness was to be found on the left side in front, but the cardiac dulness was present on the right side, and the apex-beat was in the right mammillary line. The cardiac dulness was continuous with another dull area which extended backward; for posteriorly there was a distinct dulness extending from the spine of the scapula downward and increasing more and more toward the base. In the axillary space, on the other hand, the percussion sound was tympanitic, the pitch changing at different times. High-pitched bronchial breathing was heard, with an occasional amphoric sound and accompanied by loud sonorous râles. Along its surface the right lung was held fast by firm adhesions to the parietal layer of the pleura and to the pericardium. The lung was diminished in size and showed on section a perfectly airless, hard connective tissue, beset by numerous cavities, in which the vesicular character of the lung tissue could no longer be recognized. Most of these cavities have a cylindric form and the inner surface of their walls is smooth; they correspond to enlarged bronchi the walls of which are very much thickened. No foreign body was found, but Kobler firmly believed that such a body was the cause of the disease.

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* *Deutsche Zeitschr. für Chirurgie,* VII, 1877, S. 302.

† The following exact description of the appearance of such a lung two days after the entrance of the foreign body is taken from Wagner: A bean in the left bronchus had completely occluded it. Death after two days. All the larger branches of the left bronchus were filled with grayish-red masses and grayish-yellow pus. The upper portion of the left lung was highly emphysematous and poor in blood, and under its pleura there were considerable collections of air. The rest of the lung was firm, almost airless, and the cut surface showed the finer bronchi markedly distended, either uniformly or in a sacculated manner. The lung tissue between the enlarged bronchi resembles a sponge with partitions thicker than normal and cavities much narrowed. In only a few places was the tissue air-containing; a grayish-red or grayish-yellow fluid is discharged from all parts of the lung. The alveoli are smaller than normal and the interalveolar tissue is infiltrated.

The tendency to bronchiectasis, when the foreign body is present for some time, is very noteworthy and will be referred to again later.

In the great majority of cases **septic phenomena** make their appearance; the mildest of such cases are those in which there is simple suppuration. The suppurative process is brought about by foreign bodies in themselves aseptic, through the agency of bacteria or cocci which are normally present on the surface of the bronchial mucous membrane and are enabled to pass through the epithelium after it has been injured by the foreign body. [Wood points out that if the foreign body, as a spear of wheat, breaks up, the particles migrate and infect various regions of the lung.—Ed.] Hence circumscribed pneumonia, which may result in sloughing, caseation, suppuration, or gangrene, is the commonest complication; its course depends on the variety and number of the invading bacteria and the patient's powers of resistance. [In the editor's case a child aged four swallowed a tack. A circumscribed abscess developed in the midscapular region. It terminated in spontaneous rupture into the bronchus with simultaneous expulsion of the tack. The cavity healed subsequently without event, although the child was lost sight of at the end of two months.—Ed.] A circumscribed pneumonia of this kind may be very small in extent and run its course without being recognized; or it may be multiple; or it may extend over a wide area, as we can readily understand. The subsequent course of pulmonary abscesses and gangrene originating in this way will not be discussed in this article, since chapters in our "Handbook" are devoted to these subjects. [Geo. B. Wood analyzed 89 cases of foreign bodies in the air-passages. The diseases resulting were: Gangrene, 9%, with a mortality of 75%; abscess, 29%, with a mortality in single of 30%; pneumonia, 11%, with a mortality of 40%; bronchitis, 18%, with a mortality of 6¼%. —Ed.]

A typical **bronchiectasis** not infrequently develops, and the mechanism of its production should not be difficult to understand.

If the foreign body completely occludes the bronchus, the air in the bronchial tree and in the alveoli soon undergoes absorption, admitting the possibility of collapse taking place, and atelectasis of the lung accordingly results. This is followed by a chronic inflammatory process which may last indefinitely, but in the course of this inflammatory process the foreign body again becomes movable, since a permanent hermetic occlusion is hardly conceivable. The foreign body is dislodged by ulcerative processes and, under the influence of some form of violence, is forced either upward or downward into some portion of the bronchial tree, where it descends deeper when suppuration sets in. After a time air again enters the lung and gives rise to cough, which may lead to the formation of bronchiectasis; micro-organisms also obtain free entrance and may produce suppuration or even gangrene. A combination of processes then arises which culminates in the formation of cavities, and it is impossible to say, without being accused of arbitrary assertion, whether they represent true cavities or bronchiectases.

If the bronchus is not completely occluded by the foreign body at the time of its entrance, a still greater opportunity for the formation of bronchiectasis is given; for the air can then penetrate continually into the affected side, the cough and catarrh which soon develop combine to produce a narrowing of the region occupied by the foreign body, and necessarily present some obstruction to the entrance, and still more to the escape, of the air. As will be stated once more in the section on Bronchiectasis, stenosis itself may be the ultimate cause of such formations.

How much the nature of the disease which develops as the result of the aspiration of foreign bodies is dependent on the micro-organisms that enter the body at this time has been shown by recent findings in cases of actinomycosis.* In one instance the ray fungus was present in a tooth, a fragment of which entered the lung, and a colony of actinomyces developed about the fragment of tooth. It is well known that heads and spears of grass are very often the carriers of actinomyces, and it is equally well known that these bodies or parts of them often make their way into the lungs. [Geo. B. Wood's analysis of 89 cases shows that abscess occurs in 29%, with a mortality of 100% in multiple and 30% in single abscess; gangrene occurs in 9%, with a mortality of 75%; pneumonia in 11%, mortality 40%; bronchitis 18%, mortality 6.25%.—Ed.]

In addition to these morbid processes in the lung on and around the foreign body, the development of pneumonia in the lung which does not contain the foreign body should also receive some attention; in this connection the cases of Mackay and Masius may be mentioned. I am inclined to interpret these cases as accidental coincidences. If one lung becomes atelectatic, I believe that the other as a necessary consequence undergoes dilatation; that is, it becomes swollen and more vascular. Inflammation, however, develops only when a special opportunity is given for infection. Under ordinary circumstances the compensatory powers of the individual are aroused, and the sound lung puts itself into condition to satisfy the needs of the organism. Attention has been called by Gross and Kobler to the occurrence of pleurisy in connection with the bronchopneumonia which was present in the lung.

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* Israel, *Archiv f. klin. Chir.*, vol. XXXIV; Illich, *Beitrag der Klinik der Aktinomykose*, Vienna, 1892.

Simple pneumothorax was observed in Ast's case, which strangely enough ended in recovery. More commonly the foreign body gets into the pleuræ by a process of suppuration; an encapsulated abscess results, and the prognosis becomes extremely grave. True pyopneumothorax is a pathologic curiosity.

DIAGNOSIS.

It follows from the foregoing explanation that the diagnosis may be quite simple under favorable circumstances, when a positive history is obtained and the symptoms are typical; and, on the other hand, that it may be surrounded by the greatest difficulties. It is not necessary to repeat the diagnostic points seriatim, since they have been fully discussed under the head of Symptoms. Every one of the symptoms that have been mentioned, including the rarer ones, must be thoroughly known and receive due consideration, for a single symptom may be so distinct that it alone suffices to enable one to recognize the condition. [In every case the chest should be examined by the x-ray method. It must be examined at various angles. It is true the great distress often renders the examination difficult and lessens its value.—Ed.] The most difficult cases are those in which no history is given, as the patient himself has no suspicion that a foreign body has entered his air-passages. In all such cases, which from the very outset impress the physician as diagnostic riddles, he should consider the possibility of the presence of a foreign body, especially if one of the following conditions is present: (1) Circumscribed bronchopneumonia and bronchiectases, more particularly in the lower lobe of the right lung; (2) symptoms pointing to abscess formation, when no cause for the same can be found; (3) advanced age, especially when combined with a peculiar condition of somnolence (as observed in the cases of Hamburger and Hecker) which appears inexplicable and is accompanied by marked symptoms referable to the respiratory apparatus.

In practice numerous examples are met with in which the presence of a foreign body was mistaken for croup, and vice versa; operations have even been performed on the strength of such a mistaken diagnosis. In very acute cases of asphyxia, when laryngoscopy is not practicable, the diagnosis may remain in doubt for several days.

TREATMENT.

The treatment depends altogether on circumstances; there is no single method that is applicable to all cases. Three courses are open to the physician: (1) Operation; (2) position, with the administration of emetics to favor the expulsion of the foreign body by retching and vomiting; (3) expectant treatment. These three possible methods of treatment are not always to be used separately; a combination of them is often more advisable. The attending physician should adopt a certain line of treatment after carefully weighing the individual features of the case. To lay down rules that would satisfy all the possibilities which might arise is simply impossible, and would result in an absurd schedule. By studying the case-histories contained in the literature and mastering all the possibilities of the individual methods of treatment, the physician may put himself into condition to act promptly and scientifically in any given case. There are many points in favor of operation. The glottis, as is well known, is the chief obstacle to the removal of the foreign body. Whenever the latter is forced up into the trachea by coughing, and comes in contact with the mucous membrane on and around the lower surface of the true vocal cords, a convulsive attack immediately results, the glottis closes, and the foreign body is unable to escape; on the contrary, violent attacks of coughing are produced and the patient is in danger of choking. Debilitated individuals may succumb to an attack of this kind. Low tracheotomy at once removes the chief obstacle to the escape of the foreign body and a danger which directly threatens the patient's life; it therefore satisfies two of the most important indications, and all that can be said against it is that there are a great many cases in which the foreign body succeeds in passing the glottis during an attack of coughing, that such a possibility therefore exists, and would make the operation superfluous. This is an argument of doubtful wisdom, however, for it would mean that in three-fourths of the cases all medical aid should be refused; whereas tracheotomy distinctly increases the chances of recovery and lessens the immediate danger from the foreign body, especially in children and very old people. The operation is therefore indicated in all cases in which the foreign body is movable; although attempts may first be made with position and the administration of emetics until the preliminaries for the operation can be completed. [Prolonged instrumentation favors the occurrence of pneumonia, so that Weist held that one death took place in 3½ cases not operated on, while one in 4 cases operated on terminated fatally from inflammation of the lung.—Ed.] If the foreign body is wedged fast, either in the lower portion of a main bronchus or in one of its ramifications, tracheotomy offers less chances of success, since it is not permissible to grope about in the dark with forceps and other instruments. It is true that Landgraf has shown that the bronchus may be catheterized with a suitable catheter through the mouth, and a number of cases have been reported in which the foreign body was dislodged or extracted by means of instruments introduced through the tracheal wound. I believe, however, that an attempt should always be made to obtain a view of the foreign body with the mirror introduced into the tracheal wound, and that forceps should be introduced only when the object can be distinctly seen. If it cannot be seen, any violent interference in the dark is unjustifiable; at most an oiled feather may be introduced in the hope of rendering the object movable and exciting cough so that it may be expectorated or at least brought into view.

If these methods fail, expectant treatment should not be pursued too long. Some enterprising operators have entered the mediastinum through the anterior wall of the thorax and opened the bronchi directly; but these are desperate procedures, which, all things considered, can only be justified by very unusual circumstances, and I, who am no surgeon, am not in a position to discuss them. The literature does not contain any cases successfully treated in this way. [Willard and Matás venture to hope that better results will be secured either by an anterior or a posterior route to the bronchus, providing surgeons use the Fell-O'Dwyer apparatus to keep up artificial respiration.—Ed.]

Operation has been accorded the first place because it is an extremely important and useful procedure, and must be resorted to in the great majority of cases. I distinctly emphasize, however, that it is not indicated under all circumstances, although there are radical fanatics who make this claim. These cases require a careful, conservative, and deliberate surgeon. He who is anxious for the operation, who cannot stop, cannot wait, who sacrifices everything to the one purpose of getting out the foreign body, may at times sacrifice to this object the life of his patient.

Position.—The simplest method, and the one which should be tried immediately on the occurrence of the mishap, is the laying of the patient in a suitable position. Children may be held up by the legs, and by frequent blows between the shoulder-blades the foreign body may be shaken, so that the stream of air may force it out. The fear that the foreign body, on its way out, may obstruct the glottis and cause death by suffocation is only to be considered in the case of very unfavorably formed bodies and in very weak individuals, especially old people. An adult should be laid crosswise on the bed, and, with the upper part of the body hanging down and his hands resting on the floor, should be caused to cough and retch, or he may be placed in an exaggerated Trendelenburg position. One should insist that inspiration be cautious and gradual, expiration sudden and violent. In the case of Brodie, the patient was laid on his back. I would consider the abdominal position better; for, if the head is inclined backward, the spinal column must force the trachea forward at the level of the fourth and fifth cervical vertebræ, and by this means cause a bending which is decidedly undesirable. The position of the neck should evidently be quite similar to that prescribed for tracheoscopy, which includes slight forward movement of the nape of the neck. The patient must not let his head hang forward with his chin resting on his chest, as this would compress the trachea anteriorly.

These attempts are to be made immediately, as in the beginning the foreign body is rarely very firmly fixed, and success obviously depends on its mobility. In so doing, however, it is not to be forgotten that while such bodies may be made movable again by coughing and respiration, they may also be driven further into the bronchi by such exertions if size and shape are suitable. The movements of a head of grain evidently depend on the way in which it entered. If the upper end was the first to enter, it will not penetrate deeper; if the lower end came first, it will be driven deeper by every effort made to dislodge it. It is advisable to supplement the above-mentioned methods by the use of emetics, particularly as we have in apomorphin such a pleasant and effective remedy. The use of other internal remedies cannot, in my judgment, be considered.

Expectant Treatment.—The third method of treatment still remains to be considered—namely, to wait and leave the course of events to nature. This course is naturally adopted very unwillingly by both laymen and physicians, yet it is under certain circumstances the only one, especially when the foreign body lies firmly embedded in a small branch of one of the bronchi. We may then console ourselves by the thought that the object may again become movable by inflammatory softening of the surrounding tissues, when active efforts may be made for its removal. In regard to the possible consequences, we have to consider whether they can be prevented and whether they indicate any further interference. This question will not be discussed at this point. In certain cases recovery can be expected only from the formation of an abscess and evacuation of the pus by expectoration; this applies particularly when a head of grain has gotten into the air-passages; in these special cases nature is undoubtedly the best healer, if the removal is not immediately brought about.

Among the simpler methods of treatment we find breathing with Waldenburg's apparatus recommended. The patient must, of course, be made to inspire carefully under ordinary pressure and then expire into the apparatus under negative pressure. This treatment is by no means to be despised, although I believe that it acts much too slowly and ineffectively to be applicable in ordinary cases. At times, when the physician must wait and can do nothing for a time to relieve the patient, such mild measures are invaluable. As the significance of the method is chiefly psychic, it may often be replaced by other inhalation methods or by medicinal measures.

One or two points of especial importance in the treatment may be gleaned from the statistics. It is often said that the literature is enormous; as a matter of fact, there is a distinct dearth of well-described cases that can be utilized for purposes of analysis. A more extensive collection would be of the greatest value.

We can see from the material now at hand how great is the difference in the therapeutic outlook in different cases, the difference depending in great part on the nature of the foreign body. As the physical peculiarities of the object play so important a part, I have made the classification before mentioned.

The first group, under which I have placed the objects which, on account of their hardness and smoothness, seem especially fitted for expectoration, if the location is favorable, is much too small to enable us to draw any conclusions. There are ten cases. One untreated case ended fatally; nine were cured; of these, six were tracheotomized, but in three cases the operation was without influence on the result.

We can say, then, that in these cases the outlook is favorable for the patient, and that tracheotomy may perhaps be omitted if the patient is placed in a suitable position. The ideal case is that of Niemeyer, in which expulsion was directly due to the hardness and smoothness of the bullet. I believe that the outlook in these cases may be made much more favorable by the use of cocain (without tracheotomy).

In the second group are collected those bodies which are hard and at the same time so irregular that impaction can be predicted almost with certainty. Their expulsion is possible only by means of artificial help or through the formation of an abscess, with the pus of which the objects may be expelled (51 cases). In untreated cases the chances for recovery and for death are about equal—17:21; in those cases in which the operation was performed the outlook was somewhat better—3 : 10; but the total number of cases is evidently too small. The cases are also of very unequal value.

Heads of grain constitute the next group. The chances of the spontaneous expulsion of these bodies are extremely slender. In the 16 cases reported, there was not one in which the foreign body was immediately expectorated; after a longer time (fifty days to many years) only four were expelled; 4 cases ended fatally; the rest (8) ended in recovery by means of abscesses which ruptured through the wall of the thorax. The outlook for tracheotomy seems very bad, as no operative case is found in my list. Yet as the technic advances, we shall prefer operation to the long sickness, with all its doubtful chances, when we can see the head of grain. It is worthy of note that the danger to life is no greater (1:3).

The group of fruit-seeds is, next to the second group, the greatest (43 cases). There were 22 fatal cases, against 15 recoveries. Of the recoveries, 7 were with, and 8 without, tracheotomy; while of the fatal cases, 6 were with, and 16 without, tracheotomy. The prognosis is unquestionably more unfavorable than in the two other groups, and the results speak most decidedly for tracheotomy, if conclusions may be drawn from so small a number of cases. The idea of distinguishing those that swell in water from those that do not, is useful, as the behavior of the two must be quite different. The hard ones approach in their characteristics the smooth, metallic, and glass bodies, and might very appropriately be grouped with them. Those that swell in water, however, must form a special group (30 cases). Of these, 17 died and 13 recovered. Of these cases, 28 may be still further analyzed: 11 were tracheotomized, of whom 3 died and 8 recovered; 17 were not tracheotomized, of whom 12 died and 5 recovered. Hence if we consider separately the group consisting of seeds which swell, it appears that the prognosis for the patients who are not tracheotomized is much more unfavorable, and the operation is therefore more emphatically indicated.

It is to be remembered that these bodies as they swell up become firmly embedded and cannot then be expelled. In many cases we succeed in making them movable by tracheotomy. In this, however, great caution is to be used; if the foreign body cannot be seen, the bronchi must not be explored with hard instruments, unless the surgeon wishes to invite the reproach of rough and imprudent management. [Willard states that voluntary expulsion "of seeds, etc.," takes place in 74% with recovery.—Ed.]

The last group gives no cause for remarks; as the cases present altogether too many differences, no conclusions can be drawn from such a small collection.

[Dr. Hodge has very kindly collected the cases reported since the publication of the original article and added them to the tables. Following the author's classification, the added cases yield the following percentages of deaths and recoveries. It may be said that Wood found a mortality of 34% in all cases. Spontaneous expulsion took place in 51 out of 89 cases. In only three out of 53 cases ending in complete recovery was the foreign body in the lung; while in 24 out of 30 cases that terminated in death the foreign body was not expelled or removed. In 62 of the 89 cases the foreign body was removed or expelled, and of these only 6 resulted fatally. Hence removal of the foreign body gives the best prognosis.—Ed.*]

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* The following references are appended for those interested in the surgical removal of foreign bodies from the air-passages: Crile: "Surgery," "Respiratory System," 2d Ed., p. 92.—Poulet: "Foreign Bodies," 1880, vol. II, p. 22.—Weist: "Trans. Amer. Surg. Assoc.," vol. I, 1883, p. 121.—Northrup: "N. Y. Pres. Hosp. Reports," 1896, p. 132.—Milton: "The Lancet," London, March 27, 1897.—Willard: "Trans. Amer. Surg. Assoc.," 1891, p. 345, vol. IX; "Trans. Coll. Phys.," Phila., 1891, "Am. Jour. Med. Sci.," Dec., 1891.—Gaston: "Trans. Amer. Surg. Assoc.," vol. XIV, 1896, p. 465.—Bryant: "Posterior Mediastinum," "Trans. Amer. Surg. Assoc.," vol. XIII, p. 443.—Curtis: "Annals of Surgery," 1898, vol. XXVIII, p. 605.—Rushmore: "N. Y. Med. Jour.," 1891, LIV, 85.—Matás: "Trans. Louisiana State Med. Soc.," 1898; "Trans. Southern Surg. Soc.," 1899; "Annals of Surgery," 1899, p. 409.—Depaul: "Jour. de Chirurgie," 1845.—Doyen: "Technique Chirurgicale," pp. 129–133.—Tuffier: "Société de Biologie," No. 21, 1896.—Wood, G. B.: "Foreign Bodies in the Air-passages," "Phila. Med. Jour.," June, 1899.

GROUP 1.—HARD AND SMOOTH (5 CASES).

Operated = 2

	Death.	Recovery.	
Tracheotomy	0	1	
Intubation	0	1	
	0	2	
Unoperated = 3	0	2	1 result not given.

Adding old cases (10), total:

	Death.	Recovery.	
Operated	0	8	
Unoperated	1	5	1 result not given.

GROUP 2.—HARD AND IRREGULAR (44 CASES).

	Death.	Recovery.	
Operated = 20	5	15	6 secondary thoracoplastics.
Unoperated = 24	5	19	

Adding old cases:

	Death.	Recovery.
Operated	8	25
Unoperated	22	40

GROUP 3.—HEADS OF GRAIN.

No cases.

GROUP 4.—FRUIT SEEDS.

a. Not swelling in water = 7.

	Death.	Recovery.	
Operated = 4	2	2	
Unoperated = 3	1	2	Ascaris lumbricoides complicated the fatal case.

b. Swelling in water = 20.

	Death.	Recovery.
Operated = 12	1	11
Unoperated = 8	3	5

GROUP 5.—MISCELLANEOUS.

	Death.	Recovery.
Operated	0	7
Unoperated	5	4

SUPPLEMENT.

FOREIGN BODIES IN THE AIR-PASSAGES WHICH HAVE NOT REACHED THEM FROM OUTSIDE. STONES.

LITERATURE.

Poulalion: Thèse de Paris, 1891.—Fraenkel: "Berliner klin. Wochenschr.," 1889, S. 142.—Montané: "Lancet," 1887, Jun., p. 184.—Holtz: "Allgemeine med. Central-Zeitung," Berlin, 1890.—Carlyon: "Brit. Med. Jour.," 1890, Dec., p. 1474.

Parts of our bodies, changed by disease processes, cast off, and entering the bronchi, may there act as foreign bodies.

I. The most frequent of these foreign bodies are **calcareous concretions,** which come from calcified bronchial glands, perforate the bronchial wall by processes of inflammation and suppuration, and get into the lumen of a bronchial branch. Parts of the bronchi themselves, of the lungs, or of the pleuræ, may calcify, and after being cast off get into the bronchi; tumors and hydatids growing in the lungs may in part undergo the same process. Even mucus, after lying for some time in a cavity, may be changed into a calcareous stone. In some individuals the tendency to such calcareous formations is quite surprising; they often cough up small calcareous concretions.

The statistics of these peculiar cases are found in Poulalion's works. He himself (Obs. 32) observed a man who coughed up about 100 stones; in Obs. 34 there was a stone in a cavity which evidently communicated with a bronchus; in Obs. 37, stones were found in several tuberculous cavities; in Obs. 38, a case of chronic pneumonia, there was a stone in the lungs. Observations of stones which were evidently derived from the bronchi are quoted from Andral, Dufour, Hamilton, Geribont, Vidal, Leclère, and Moscato. These stones were characterized by various processes, evidently corresponding to ramifications of the bronchi. They are, therefore, often extremely irregular in form; the surface is smooth or slightly mammillated; the size is very variable, from that of a bean to that of a cherry; they vary also in consistency, some being quite friable; the color is whitish or gray. On section, layers have been found, and also fluid in the center, as in gall-stones; and, finally, foreign bodies have been demonstrated as forming the nuclei of broncholiths in some instances. The chemical constituents are, in the main, calcium phosphates and carbonates, so combined that the former usually predominate. But the stone reported by Fraenkel consisted essentially of calcium carbonate. In the analysis of Audouard, 26.3 parts of calcium phosphate to 23.4 parts of calcium carbonate were found. The stone of Montané had the same composition. Magnesium carbonate was found by l'Heritier.

The formation of these stones is as follows: the sputum stagnates in certain places, calcium deposits settle upon it, and finally so increase that a firm stone is the result. Blood-clots, membranes, and even foreign bodies may, of course, serve as places of deposit for the calcium salts as well as thickened bronchial mucus. We cannot, of course, doubt that a certain constitutional anomaly plays a rôle in the occurrence of such excretions. There are people, however, who excrete an unusually large supply of calcium through the lungs. This may be favored in a high degree by manner of life and by disease, but a constitutional tendency must play a part in it. I myself recovered a bronchial stone from a twenty-year-old girl who suffered from chronic bronchitis and whose father had a characteristic renal calculus.

(*a*) Besides the calcareous concretions, cartilaginous and bony masses have been found free in the air-passages and coughed up. Thus, the cartilages of the bronchi or of the trachea may slough off and act like free bodies; enchondromata may form in the lungs, not only in the bronchial wall, but also in the pleura, and may become free. But masses frequently occur in such places which must be described as bone, because they have true bone-corpuscles and Haversian canals. They are nearly related to the cartilaginous masses and often arise from them.

[Pollack * studied bone formation in the lungs. Of 60 lungs examined, 17% contained true bone as small, hard, yellow masses. They originate by metaplasia from scar tissue and calcified necrotic tissue. See also Ansperger,† who speaks of nodular and dendritic bone formation.—Ed.]

(*b*) Fragments of soft tissue are at times cast off in phthisis and other ulcerative processes of the lungs, and enter the bronchi.‡ After lying in the bronchi for some time, they also become calcified. All these formations cause, as a rule, almost no pathologic symptoms; they are coughed up and are accidentally found in the sputum, in which one also occasionally finds, if he searches diligently with the microscope, calcium crystals and masses of calcium, of which we as yet do not know the significance. In individual cases, however, larger concretions have caused very severe pathologic symptoms, which are quite equal to those which we have seen as a result of the inspiration of foreign bodies. A physician once coughed up such a concretion, and Sander has given us a detailed description of the case. He had for more than two years suffered with severe chest symptoms, had had several attacks of pneumonia and pleurisy, with hemoptysis, until he was finally cured by the expulsion of a stone which consisted of calcium carbonate mingled with crystals of fatty acids. Sander also cites two cases in which such concretions caused no severe symptoms, but, as usual, were easily coughed up; also two cases reported by Leclère. Poulalion has made a further collection of the material, and we may now speak of the symptoms and course of **broncholithiasis.**

As in gall-stones and renal stones, we find cases enough in which at the autopsy we are surprised at the presence of stones which had caused no symptoms at all. If a stone lies firmly fixed, if it is aseptic, if no infection enters from without, there is no reason why symptoms should be present. But if one of these conditions is not fulfilled, then acute and chronic disturbances arise, which may be combined in manifold ways, and under certain circumstances present a characteristic clinical picture. Thus, Poulalion even speaks of a "colique bronchique"; but I have no wish to introduce into our literature this piquant and, for some ears, no doubt, charming word, *bronchial colic*, and shall therefore content myself with the older word "stone-asthma."

The following symptom-complex is characteristic of **stone-asthma**:

Convulsive cough, attacks of dyspnea, and the expectoration of one or several small stones (broncholiths). The patient perceives a sense of heaviness, of pressure, finally of unendurable burning in the lower part of the trachea; cough comes on resembling an attack of whooping-cough; like the latter increases to the point of intense dyspnea, arousing in the patient the fear of impending death; and may go on to stupor. Suddenly, with a feeling of severe rending pain, the stone is thrown out, great relief is experienced, and the attack is over. In many cases, however, the attacks are abortive, the stone is not coughed up, the convulsive cough is less severe, but frequently returns. The patients may even assert at times that they have the feeling as if the stone was trying to get out, as if it moved up in the neck, but dropped back again. Such attacks may pass very quickly, but they may also last a long time; Poulalion's patient, Necker, had an attack that lasted forty-eight hours.

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* *Arch. für path. Anat. u. Phys.*, 1901.

† "Ziegler's Beiträge," vol. XXI, 1897.

‡ Holtz: A lung sequestrum [or sphacelus.—Ed.], 10 cm. long and 2.5 cm. wide, expectorated in chalicosis pulmonalis.

Some of the cases from the first assume a chronic form which often suggests tuberculosis. The earlier writers aptly described this form as *pseudo-phthisis calculosa.* In most cases bronchiectases form, or abscesses with all their attendant possibilities. In such cases the presence of the concretion is usually not suspected and is only revealed by the autopsy or by the broncholith being accidentally coughed up. In many cases, after the expectoration of a concretion recovery took place, but fatal cases are also recorded. In Leclère's case multiple abscesses developed, broke through into the pleura, and hydropneumothorax was the result. In the case reported by Dalma, a stone had almost entirely occluded the bronchus leading to the lower lobe of the right lung. To the distal side of the broncholith the bronchi were enlarged, inflamed, and filled with pus. Several of them opened into the pleural cavity, which became the seat of empyema.

Finally, there are cases in which stone-asthma passes into the chronic course of calculous diseases. All the variations which occur in such a course may be learned by reading Poulalion's observations 46, 47, and 32. From the last-named I give a short excerpt:

The patient comes of a family which is free from tuberculosis; a brother was nervous and is melancholic. He himself has had various diseases of the thoracic organs and has been a heavy drinker since 1870. In 1875 he had an attack of delirium tremens, and since then has suffered from chronic bronchial catarrh. He was first in the hospital in 1889, when he expectorated several stones. He has attacks of coughing that last two or three hours and are interrupted by only short periods of rest. They begin with a feeling of tickling in the throat and retrosternal pain. The stones are expelled during or at the end of an attack of coughing, either alone or with sputum, and then follow rest and comfort. It is rare for more than one to be expelled in one day, and often only one in three or four days. Blood was never found in the sputum. Appetite and general condition are good; urine normal. In the lungs we find only some dulness over the left apex. Tubercle bacilli are always sought in vain.

A year later he again came to the hospital in about the same condition. When he coughs numerous râles, mostly moist and bubbling, can be heard in the left axillary space. A chronic deterioration is apparent; fever and loss of appetite are increased; the stones are expectorated as before. Sometimes the expulsion of the stone is accompanied by hemoptysis. At the autopsy we find a chronic pneumonia on both sides. The left lung is adherent over the greater part of its surface. In both lungs we find concretions in small hollow spaces in the tissue. These lie in the lung tissue itself and their connection with the bronchi is not evident. Others are found in small cavities which open directly into enlarged bronchi.

[Atlee* reports as follows: Patient complaining of bronchitis; pain in left side, increasing temperature, cough, and expectoration; bronchial breathing; dulness over painful area. Expectoration by ninth day greenish-fetid. On twelfth day concretion, $\frac{3}{4}$ inch long, conical, $\frac{1}{4}$ inch diameter, containing lime and phosphorus. Recovery rapid. No tubercle bacilli. The author quotes Leroy and others, giving non-tuberculous cases of bronchial concretions. These cases may run a chronic bronchiectatic course or may resemble pneumonia. The concretions have been ascribed to (1) inspired dust; (2) calcification of glands and ulceration into bronchi; (3) long-continued irritation of bronchial cartilages in bronchiectasis, with true bone formation and detachment thereof (Leroy).—Ed.]

The **symptoms** of broncholithiasis are:

Expulsion of stones.

Admixture of blood with the sputum. This rare hemoptysis is usually insignificant and belongs to the advanced stages of the disease, yet it may be abundant and even fatal, as the case of Caseau shows.†

The stone, when it is expectorated, is frequently tinged with blood.

Pain.—Most of these patients have pains of an indefinite character, but often only a feeling of pressure on the chest. At the time of the expulsion of the stone the pains may be very severe. Only with the greatest caution can conclusions be drawn from the location of the pain. In the case of Montané the painful spot was a little beyond the left sternoclavicular articulation; the stone was in the left bronchus.

Cough occurs only during the attack and is of a violent, convulsive character; otherwise it is independent of the stone, but is never lacking in an extensive bronchitis.

Dyspnea exists with characteristic severity only during the attack; at other times it is absent or dependent on secondary changes.

Fever is due to the inflammatory process which may develop after the formation of the stone, or may be entirely absent.

Diagnosis.—The recognition of stone-asthma is possible only when a stone is expectorated or when we are dealing with a patient who has formerly coughed up such stones. Such patients often know their condition so well that they can predict the approach of an attack. When a stone has been expelled, the only question to be decided is where it comes from. Calcareous masses have been known to collect on the teeth, in the crypts of the tonsils, and in the pouches of Morgagni, and have been expelled by coughing. If coryza is present, we must think of a rhinolith, which may have come from the nasal cavity and entered the mouth. We must also think of true foreign bodies derived from the outside and entering the air-passages without the patient's knowledge.

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* *Am. Jour. Med. Sci.*, July, 1901.

† "Hémoptysie foudroyante calculeuse," *Bull. de la Soc. anat. de Paris*, 1837, vol. IX, p. 100.

The recognition of *pseudo-phthisis calculosa* is, according to the present condition of things, possible when any circumstance, especially an attack of stone-asthma, has called attention to the stone formation; in other cases all these circumstances will be regarded as chronic pneumonia, the cause of which is unknown.

No **therapeutic measures** of any value are known for this stone formation in the lungs; but it would seem rational to give an acid diet and to have the patient breathe simple acid vapors.

II. In regard to the excretion of **uric acid salts** in the bronchi in gouty individuals, I have found observations by Bence Jones ("Lancet," 1856), who saw in a gouty patient deposits of urates in the bronchial walls; and another by Bernheim ("Soc. de méd. de Strasbourg," 6 Juin 1872), who said in a discussion that concretions of sodium urate occurred in the bronchi, being expelled in the form of stones.

III. Thrush in the Bronchi.—As Virchow has already shown, thrush very rarely enters the bronchi; Schmidt ("Beiträge zur path. Anatomie und allgem. Pathologie," 1890, VIII, S. 173) has observed it in five children. The bronchial mucous membrane was covered with a whitish layer, which consisted of a mass of exudate with various cellular elements. The thrush fungus was quite uniformly distributed throughout this exudate. The fungi did not invade the ciliated epithelium, which is much more resistant than the squamous form.

IV. Other fungi that find their way into the lungs, especially the **mold fungi,** also occur incidentally in the bronchi. [In another section pulmonary aspergillosis, streptothrix infections, and other parasitic infections of the bronchi giving rise to bronchitis will be considered.—ED.]

BRONCHITIS.

Since the publication of Laennec's work considerable progress has been made in the study of the various forms of bronchitis, and it looks as if in this field also the knowledge of pathogenic organisms were destined to afford a clearer insight.

Among the comprehensive works on bronchitis may be mentioned the following:

Laennec: "Traité de l'ausc. méd."—Stokes: "A Treatise on the Diagnosis and Treatment of Diseases of the Chest."—Lasègue: "Etudes médicales," t. II, 1884.—Ferrand: "Leçons cliniques sur les formes et le traitement des bronchites," Paris, 1888.—Sée, G.: "Bronchites aiguës," Paris, 1885; "Bronchites chroniques," Paris, 1886.—Riegel: "Krankheiten der Trachea und der Bronchien," in Ziemssen's "Handbuch," Bd. IV, 2. Hälfte.—Fothergill: "Chronic Bronchitis," London, 1882.—Hamilton: "On the Pathology of Bronchitis, Catarrhal Pneumonia, Tubercle and Allied Lesions," London, 1883.—Lebert: "Klinik der Brustkrankheiten," Tübingen, 1874.—Fraenkel: "Krankheiten des Respirations-apparates," 1890.—Knauthe: Article on "Bronchitis catarrhalis" in Eulenburg's "Realencyklopädie."—Forcheimer: "Streptococcus Bronchitis," "Medical News," June 1, 1901.—Fuchs: "Die Bronchitis der Kinder," 1849.—Gairdner: "On the Pathological Anatomy of Bronchitis," etc., Edinburgh, 1850.—Hayem: "Des Bronchitis," Thèse d'agrégation, 1869.—Greenhow: "On Bronchitis," 1878.—Murrell, William: "Chronic Bronchitis and its Treatment," 1889.—Ewart, William: "The Bronchi and Pulmonary Blood-vessels, their Anatomy and Nomenclature," London, 1889.—Barth and Blachez: "Maladies des bronches," "Dict. encycl. des sc. med.," 1869, 1ère, vol. v.—Hamilton, D. J.: "Text-Book of Pathology," vol. II, p. 72, 1894.—Clark, Sir Andrew: "The Barking Cough of Puberty," "Med. Soc. Trans.," vol. XIV, p. 142, 1891.

Classification.—Owing to the number of morbid conditions which here claim our attention the first requisite is a useful classification.

Ferrand divides bronchitis into the following forms: (1) Congestive, (2) catarrhal, (3) inflammatory, (4) spasmodic, (5) infectious. While his theoretic explanation of the differences between congestive catarrh and inflammation are worthy of attention, the classification cannot be utilized in practice. For my part, I can never tell at the bedside when catarrh or inflammation begins. We use the word bronchitis without troubling ourselves to inquire whether the amount of secretion is increased, whether it is normal in character and amount, or whether alterations, especially of the morphologic elements, can be demonstrated. Ferrand bases the diagnosis chiefly on the sputum; in the congestive form the sputum is extremely tenacious and scanty; in the catarrhal form it is abundant, mucous, and fluid; in the inflammatory form, purulent and mucopurulent, and variable in amount.

It appears, therefore, that he divides into three groups events which are quite usually observed in succession in the same patient. This may be justified by the fact that in numerous cases only one of these forms develops, and then becomes permanent. If in these cases we speak of dry catarrh (*catarrhe sec*) or of mucous discharge (*catarrhus pituitosus*), while Ferrand uses the terms congestive and catarrhal bronchitis for the same conditions, the difference is merely verbal. There is one objection to the use of Ferrand's terms: they contain an explanation which is not always appropriate and necessarily varies with the hypothesis, while our old purely descriptive terms will retain their usefulness even when we shall have come to know every form of bronchitis, both etiologically and pathologically, in all its details.

Riegel, whose treatise on bronchitis is the standard in Germany, distinguishes: acute tracheobronchitis; acute catarrh of the secondary and of the tertiary bronchi (*bronchitis capillaris*); and chronic bronchitis, the latter being further subdivided into a dry form, a mild form with abundant mucous discharge, bronchoblennorrhea, serous bronchorrhea, and putrid bronchitis. G. Sée, who is by many regarded as an authority, should be followed with great caution, as he grasps at any point of view which strikes him as clever, and loses himself in speculation. Hence his description of the disease, although minute, is neither exhaustive nor logical.

Fothergill, in his "Chronic Bronchitis," the second edition of which appeared in 1889, views the question from a thoroughly practical standpoint. He gives an excellent description of the condition of certain old bronchitic subjects, discusses the symptomatology, and reviews the relations of bronchitis to bronchiectasis, emphysema, and tuberculosis. He then distinguishes the following forms: dry, asthmatic, catarrhal, cirrhotic, emphysematous, degenerative, mitral, and gouty bronchitis. The last chapter is devoted to treatment. I have not gained any essentially new points of view from the reading of his book, but some of his remarks will appear at the appropriate places.

Among the books to which one naturally turns when in perplexity, I would name Walsh's "Practical Treatise on the Diseases of the Lungs." This author divides them into:

1. Acute bronchitis: (*a*) of the primary and secondary; (*b*) of all the bronchi, including the capillary;
2. Chronic bronchitis;
3. Special varieties: (*a*) fibrinous; (*b*) mechanical (scissors-grinders' and coal-workers'); (*c*) stone-asthma; (*d*) acute specific forms (typhoid, syphilitic); (*e*) diathetic forms; (*f*) secondary forms in thoracic diseases.

The superiority of this classification to that of Fothergill is at once apparent. Excepting that of Riegel, it is the only one that can be considered in actual practice.

The attempts to establish a distinction between catarrh and inflammation of mucous membrane have now been abandoned and the two words have the same meaning. They indicate processes which imperceptibly merge into one another from the mildest to the severest forms and are characterized by pain, hyperemia of the mucous membrane, the presence of a secretion containing more or less cellular constituents, and by an entire lack of any tendency to ulcerative or destructive processes: if small ulcers do occasionally form, they heal very easily. If the disease is protracted, it may lead to atrophic or hypertrophic processes in the mucous membrane, according to its nature, but it is never destructive.

We distinguish many different kinds of bronchitis, but the clinical pictures are so ill-defined that they readily merge into one another and often give rise to doubt. Viewing the matter from a purely practical standpoint we have, *first,* those forms which occupy an independent and clearly defined position in nosology and probably represent characteristic diseases. These are the following: simple acute bronchitis or tracheobronchitis; acute capillary bronchitis; chronic bronchitis; putrid or fetid bronchitis; fibrinous bronchitis.

Next to these we have those forms which merely accompany other diseases and play quite a secondary part, or at least rarely predominate in the clinical picture.

There are cases in which the tracheal symptoms are so conspicuous, and so completely dominate the clinical picture, that we have the right to speak of a trachitis; but the trachea is always involved to a greater or less extent in any form of bronchitis, and the term tracheobronchitis would therefore be quite justifiable if it were not so unwieldy.

Another classification divides the various manifestations of the disease into ascending and descending forms. The former are secondary to catarrh of the nose and larynx, the latter follow inflammation of the lungs. In the second group the pulmonary phenomena dominate the clinical picture to such an extent that the bronchitis in comparison becomes quite insignificant. The first group, on the other hand, is of great importance, and is often met in practice among benign catarrhal forms of bronchitis.

The total depravity of systematic writers is shown by the fact that bronchitis has been divided into a form with moist râles and one with dry râles.

Such expressions as asthmatic or emphysematous bronchitis will no doubt appeal to the average practitioner, for it is often impossible to tell whether the catarrhal or the asthmatic element predominates in the clinical picture. I am afraid that for similar reasons we shall soon find the literature encumbered with such terms as "asthmatico-emphysematous bronchitis," "broncho-emphysematous asthma," "asthmatico-bronchitic emphysema," and the like.

What shall be said of a form which is known as mitral bronchitis? There are undoubtedly cases in which bronchitis is an accompaniment of mitral disease, and there are cases of mitral disease accompanying bronchitis. It is possible that both conditions may have a common origin, such as gout or some other constitutional anomaly. The term mitral bronchitis represents certain pathologic findings, and is undoubtedly dictated by an effort to distinguish a special clinical picture from others; to that extent terms of this kind have a certain practical value, but they are made for private use and are often based on a comparatively small number of observations. They should, therefore, not be used too freely; most of them are ephemeral and merely reflect a momentary point of view for which a clever author has succeeded in winning temporary acceptance.

An etiologic classification is unquestionably to be preferred to any other, although at the present time it is still hampered by the fact that our knowledge is unfortunately very incomplete. Marfan has given us a classification of this kind which corresponds as closely as possible to the present state of knowledge. He distinguishes:

Specific bronchitis: (*a*) germs entering from without: bronchitis in the course of influenza, whooping-cough, measles, diphtheria, pneumococcus infection, erysipelas, after the inhalation of anthrax, in tuberculosis, thrush; (*b*) germs entering through the blood: bronchitis occurring in the course of variola, malaria, glanders, and secondary syphilis.

Non-specific bronchitis: bronchitis following cold; in chronic diseases of the nasopharynx; in chronic affections of the lungs, heart, and mediastinum; in angioneurosis (hay-fever, bronchial urticaria); in asthma; in arthritic affections; in gout; in dilatation of the stomach and in the lymphatic diathesis; in diseases of the blood-vessels; in albuminuria; in adynamic conditions (protracted fevers, cachexia, moribund conditions); in elimination intoxications (iodin, bromids, cantharides); after the inhalation of noxious dust and gases.

This classification obviously presents many weak points. The boundary-line between specific and non-specific bronchitis is arbitrary and depends altogether on the state of bacteriologic knowledge. [And yet such classification is practical and will in time prevail. The various micro-organisms give rise to forms of bronchitis which differ in course, duration, and degree of severity. A streptococcus bronchitis with definite features undoubtedly prevails. Bacteriologic methods must be employed to confirm our clinical observations.—Ed.] I therefore prefer to give a simple enumeration of the known forms, based on our knowledge of etiology, without attempting any doubtful subdivisions, and shall merely add a few necessary remarks about each form. In this way all that is known of the etiology of bronchitis will be presented in the simplest form.

FORMS OF BRONCHITIS.

1. Acute bronchitis due to unknown injuries. Bronchitis from catching cold.
2. Bronchitis due to the inhalation of dust.
3. Bronchitis due to the inhalation of gases.
4. Bronchitis due to specific infection of the surface of the mucous membrane: whooping-cough, erysipelas, diphtheria, and putrid bronchitis.
5. Bronchitis due to nervous disturbances.
6. Bronchitis occurring in constitutional diseases.
7. Bronchitis occurring in infectious diseases*: measles, smallpox, typhoid fever, intermittent fever, glanders, syphilis, tuberculosis, relapsing fever.

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* The dividing-line between this group and group No. 4 is in many cases arbitrary, as, for instance, in glanders and in tuberculosis.

8. Bronchitis occurring in the course of intoxications.

9. Bronchitis occurring in disease of the upper air-passages and of the lung, pleuræ, mediastinum, and thorax (kyphosis and scoliosis).

10. Bronchitis in diseases of the heart and blood-vessels.

11. Bronchitis in diseases of the digestive organs.

12. Bronchitis in diseases of the kidneys.

13. Bronchitis in diseases of the skin.

[Fowler and Godlee's * classification is as follows:

I. Acute bronchitis (*a*) of the larger tubes; (*b*) of the smaller tubes (capillary bronchitis).

II. Chronic bronchitis.

III. Secondary bronchitis: (*a*) in acute febrile diseases; (*b*) in gout; (*c*) in tuberculosis; (*d*) in syphilis.

IV. Plastic bronchitis (a separate disease, the authors assert).—Ed.]

ETIOLOGY.

After this summary review of what is known about the etiology of bronchitis (which will be still further elaborated in the special articles), an explanation of the origin and mode of extension of catarrh in general is desirable. But with the tendency to discredit general views and speculations and to limit the field of medical investigation to the exact representation of facts, vague dissertations about the influence of wind, moisture, and changes of temperature,—in other words, all the conceivable influences which the external world exerts upon man,—have fallen more and more into disrepute.

["Heredity and temperament constitute distinct factors; a delicate mucous membrane may be inherited, as a delicate skin or any other outward peculiarity may be" (Ewart).—Ed.]

There is no object in speaking of the geographic distribution of bronchitis, as there are no available statistics. To say that it is admitted that, *ceteris paribus*, catarrhal affections of the respiratory organs increase in frequency the further we go from the tropics toward more northern latitudes is, after all, only a truism which in general has a certain degree of probability. It would no doubt be very useful to know definitely that the danger of acquiring catarrh was peculiarly great in this or that place. Hirsch, who is the most experienced authority on this subject, asserts that numerous observers testify to the rarity of bronchitis in Egypt, on the plains of India, on the western plains of America, in California, and in portions of the Antilles; he also says that mountainous countries and high plateaus are frequently the home of catarrh. Lebert has also discussed at length the meteorologic influences. In Germany bronchitis, as a rule, increases as autumn approaches, reaches its highest point during the winter, and diminishes in the spring. Bad weather also increases the tendency to bronchial catarrh.

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* Fowler and Godlee, "The Diseases of the Lungs," London, 1898.

The discussions in regard to the individual predisposition are equally commonplace. Strong individuals are less disposed, while the weak are more so; yet surprising exceptions occur, etc. From this it follows that childhood is especially predisposed; at this time acute catarrhs occur very frequently, while the tendency to chronic catarrhs seems greater in old age. These speculations are of little value, and the investigation of special relations will, I hope, relieve us more and more of these distressingly axiomatic generalities.

A few remarks should be added to the above-mentioned etiologic subdivisions:

1. The discussion of "**cold**" as the cause of certain inflammations, which are generally characterized as rheumatic and show a preference for certain mucous membranes, occupies a large place in the literature. Although the controversy has produced little that is of any value, it must nevertheless be mentioned here. We catch cold because the body or a part of it suffers a relatively sudden deprivation of heat and fails to react as vigorously as it would under normal conditions. The human body is often exposed to abrupt changes of temperature without suffering harm, as it possesses in the circulation and in the contraction of the smooth muscles of the skin an effective means of protecting itself against the danger of sudden variations of temperature within a relatively wide range. But the nervous system is not always sufficiently vigilant, the mechanisms which regulate these changes are not always energetic enough, and hence injuries result. These matters are easily understood; Riegel has explained them in connection with a Rosenthal lecture in Ziemssen's "Handbuch."

Rossbach * has recently experimented on the influence of cold, and on the application of cold to the skin. He saw the blood-vessels of the trachea contract reflexly after one to two minutes; this was soon followed by a venous hyperemia and an increased secretion of mucus. This experiment, while it shows the powerful reflex influence of the skin on the mucous membranes of the air-passages, obviously does not explain the catarrh.

The injurious influence of *moist cold air*, when the temperature changes suddenly through several degrees and the change is accompanied by a strong wind, is a matter of such universal experience that it must be accepted as a fact.

The question is no longer whether cold can injure, but in what manner it works the injury. If the action of the vasoconstrictors and vasodilators of the different blood-vessels of the skin is disturbed even for a short time, the whole body will be affected; perhaps the heat production is disturbed, or perhaps a hyperemia of the internal organs is favored. But it is still uncertain why a bronchitis should result more than some other injury; yet it is such a common occurrence that few men will be willing to give up the idea of its being directly caused by catching cold. Even if the clearest theoretic deductions showed it to be untrue, most men would still exclaim: "Yes, but don't you see I have caught cold."

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* "Festschrift zur Feier des 300jährigen Bestandes," etc., Leipzig, 1882.

At present we are inclined to go to our bacteriologic experiments for an explanation: The air-passages always contain micro-organisms; in the great majority of cases they contain micro-organisms that we know are capable of causing inflammation, especially Staphylococcus pyogenes, Streptococcus pyogenes, Fraenkel's pneumococcus, and Friedländer's pneumococcus. In healthy men these micro-organisms remain in the bronchi without doing any injury, as the well-preserved epithelium covering the membrane is sufficient to prevent the entrance of the germs and to force them out. Investigations on this point, in spite of their extensiveness and the industry of their authors, are not yet sufficient, as the work is so great. Pansieri finds in the healthy bronchi 8 different kinds of streptococci, several kinds of sarcinæ, 21 kinds of bacilli, and 10 kinds of micrococci. As soon as the protecting epithelial covering is broken through, the micro-organisms which are present at that point effect an entrance and immediately excite a circumscribed inflammation. [Hence to infection as the exciting or active agent we must attribute most cases of "cold" bronchitis.—Ed.] The body exerts all its powers to prevent the entrance of these organisms; how far it succeeds in this effort depends on the extent of the loss of epithelium, and upon the powers of resistance which are at hand. In pharyngitis any one can observe for himself how at first a small spot becomes reddened, and how from this spot the inflammation spreads until in twenty-four to forty-eight hours the entire throat is involved. When the epithelium is loosened, it can no longer resist the attack of the microbes from the side and from below; this duty evidently devolves on wandering cells which come from the blood-vessels, fill up all passages, and check the advance of the process. They succeed best in preventing the downward extension, since they start from below in response to the external irritant; at the sides, however (*i. e.*, on the surface), the loosening and desquamation of the epithelium go on until a physiologic boundary is reached which checks the desquamation; a boundary of this kind is found wherever one kind of epithelium merges into another. Thus, an angina does not easily extend into the larynx; a urethritis does not easily give rise to a cystitis, nor a catarrh of the cervix uteri extend to the body of the uterus. In many places the boundaries are evidently so fixed that it is really exceptional to see the inflammation cross them; as, for example, between the external skin and the mucous membranes, the esophagus and stomach, etc., although of course there is no region that is hermetically sealed.

So, in taking cold, we can suppose that there is a loosening of the epithelium of the bronchi, with the development of small thin places, in consequence of sudden changes of temperature in people who are not sufficiently hardened. As a result, dust particles and micro-organisms penetrate into these thinner places and cause a desquamation of the superficial layers of the epithelial covering, which otherwise close the tissues hermetically; this layer must then be newly formed from below. This, in general, is the process which we call catarrhal inflammation. But this is, of course, a general statement that does not apply only to catarrhal inflammation; specific cocci of some kind may also penetrate into these exposed places, and we have then a specific inflammation, such as erysipelas or diphtheria. Many of these cocci are perhaps pathogenic in so high a degree that they are capable of penetrating an epithelium which is quite sound, if they only reach it from without. We can always understand that taking cold brings about conditions which are favorable to the beginning of every kind of inflammation of the mucous membranes. As the mucous membranes have different powers of resistance according to the habits and manner of life, it is clear that the same injurious influences that are borne quite easily by one may cause severe illness in another. How far this may extend depends also on the toxicity of the micro-organisms which enter and upon the energy with which the protective cells and exudates arise to check the penetration.

2. In addition to cold, **dust inhalation** is to be considered as a quite common injurious factor which disposes to different forms of bronchitis. It is certain that every kind of dust is injurious, but it is just as certain that the amount and kind of dust make a great difference. This cannot be so well studied in ordinary life, where we have to do with complex and very variable conditions, as among workers in dust; that is, people who by their occupation are compelled to breathe continuously certain kinds of dust. Such cases present excellent examples of certain kinds of chronic bronchitis. But, besides this, we must remember that dust, if present continually in any lung, acts as an enemy, which just as frequently, if not more frequently than cold, threatens the continuity of the protective covering. It is, in fact, marvelous that these delicate epithelial structures offer so long and so energetic a resistance to the dust. That in a ballroom the dust reaches even to the bronchi is not to be doubted, although so much is intercepted by the mucous membrane of the nose; speech favors the introduction too much for any doubt to be possible. Our interest is much more aroused by those occupations in which the workers are continuously exposed to a dust-laden atmosphere. After the loud explosions in the mines such a quantity of very fine dust is found in the air that it may penetrate and occlude the finest bronchi. Yet it is certain that these dangers may be easily exaggerated. These workmen can always work with their mouths closed, and most of the dust is thus intercepted by the protective apparatus: if the people were not so usually alcoholics, if they did not so frequently sin through ignorance as they do, then the dust would not be so dangerous as it is to-day in spite of all hygienic precautions. The works which are occupied with the diseases of working-people give most detailed descriptions of these matters. Vegetable dusts and mixed dusts are most injurious in their influence; then follow the metallic, then the animal, and lastly

the mineral. According to Hirt, who supports his statement by observations on more than 12,000 dust-workers, of 100 who were attacked with chronic catarrh: (1) 14.8 worked in metallic dusts; (2) 11.0 worked in mineral dusts; (3) 19.0 worked in vegetable dusts; (4) 13.6 worked in animal dusts; (5) 18.4 worked in mixed dusts.

The value of statistics of this kind is extremely slight, since in each group different conditions and classes are included.

Under 1 are included engravers, painters, watch-makers, tinkers, file-cutters, coppersmiths, grinders, engravers, pin-workers, bookbinders, lithographers, knife-, nail-, and artillery-smiths, braziers, zinc-workers, sieve-makers, blacksmiths, brass-founders, dyers, locksmiths, varnishers, needle-makers, gilders, brocade-workers, workers in certain paper fabrics, and letter-founders.

Under 2, flint-stone workers, millstone-workers, house-painters, agate-polishers, porcelain-workers, ultra-marine workers, soapstone workers, polishers, potters, slate-makers, masons, diamond-workers, cement-workers, workers in the stamping of glassware, and lithographers are included.

Under 3, millers, coal-handlers, weavers, chimney-sweeps, bakers, confectioners, cabinet-makers, carpenters, wood-turners, saw-mill-workers, rope-makers, upholsterers, coal-miners, and cigar-makers.

Under 4, brush-, cotton-, and flax-workers, workers in certain paper fabrics, horsehair carders, comb-makers, horn and bone workers, hair-dressers, paper-hangers, furriers, turners, saddlers, button-makers, hat-makers, cloth-makers, and sewing machine operators.

Under 5, glass-cutters, glaziers, street-sweepers, day-laborers, and certain hat-makers.

Such statistics give only a general impression; it is easy to see, for instance, how improper it is to group together millers and confectioners, and to what a variety of dangers workers in the same trade even are exposed. Many dangers have disappeared with the advance of industrial methods, but the opposite is also true. According to Merkel, the following anatomic and clinical varieties of dust-diseases have been observed up to the present time: (1) Coal-dust, soot, and graphite; (2) ferrous oxid, ferric oxid, and ferric phosphate, mixture of steel and sandstone dust (grindstone); (3) stone dust and clay dust; (4) tobacco dust; (5) cotton dust.

Many workmen, in whom the entrance of dust into the air-passages is undoubted, are exposed to such a complication of injuries that the dust plays but an accessory part in the development of their thoracic diseases.

[Strongly irritating particles or vapors may act as direct exciters of bronchitis, as, for instance, the vapor of ammonia, of iodin, of bromin; finely divided ipecacuanha, pepper, or tobacco; and, in the case of those especially liable, the pollen of certain varieties of flowering grass. So-called hay-asthma is a striking example. It is caused by the inhalation of *Anthoxanthum odoratum*. Violent and continued sneezing, dyspnea occurring in paroxysms, oppression and retrosternal soreness, and wearisome cough, which is at first dry but ultimately may produce a varying amount of watery, mucoid, or faintly opaque expectoration, are the main symptoms in cases involving the bronchi (Ewart).—Ed.]

3. **The Inhalation of Gases.**—In this class also occupation-diseases claim the largest share of our interest. The following substances are to be especially noted: Nitrous and nitric acid, pure nitric acid, which is relatively least dangerous, sulphuric acid, chlorin gas, muriatic acid, iodin, bromin, oil-vapors, lime-vapors, and the vapors arising from burning tar.

Amyl alcohol and amyl ether ($C_{10}H_{11}O$) exert so powerful an influence on the mucous membranes that they excite violent coughing and even expectoration of blood.

Inhalation of carbonic acid also causes cough and catarrh of the mucous membranes. The amount of carbonic acid in the air of a dwelling-room should never exceed 1 per thousand. On the other hand, illuminating gas appears to be harmless.

Ammonia and chlorin excite an inflammation which, with expectoration of blood, may go on to croup and pneumonia. The vapors of muriatic acid are similar in their influence to those of chlorin. Sulphuric acid also causes a very decided bronchitis; that excited by nitric acid is still more intense, presenting all the symptoms of an increasing bronchitis, and sometimes terminating in edema of the lungs and death. It is well known that attacks of asthma are treated by the inhalation of the vapors which arise from the burning of niter paper. This, however, generates not nitric acid, but ammonia and carbonic acid. The stable decomposition products of the niter are mechanically separated as potassium carbonate and potassium nitrate.

Hydrogen sulphid is not dangerous to the bronchi; neither is carbon disulphid. In the same way hydrogen arsenid, hydrogen antimonid, and hydrogen phosphorid, while they are fatal to the organism if they enter the blood, do not cause any recognizable injury in the bronchi; on the other hand, hydrogen silico-fluorid and hydrogen selenid have an irritating influence on the mucous membrane.

The inhalation of the vapor of concentrated nitric acid causes relatively little irritation and excites only a hyperemia of the bronchial mucous membrane; the substance has even been recommended for medicinal use in the form of inhalations! Among workers in melinite factories attacks of dyspnea with dry cough, accelerated respiration, anemia, and great weakness have been observed; these attacks are caused by the picric acid which is developed during the work. Reynault and Sarlet have described this condition as *bronchite méliniteuse.**

4. **Infection of the Surface of the Mucous Membrane.**—It is supposed that specific germs reach the bronchi through inhalation and

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* *Annales d'hygiène publiques*, XXV.

cause peculiar diseases if they find a place to establish themselves. The healthy mucous membrane is unquestionably a very poor medium for the growth of micro-organisms; hence predisposing factors must be present to facilitate the entrance of the germs. Infection with the tubercle bacillus has never been satisfactorily explained. Can it take place through the normal bronchial mucous membrane?

[Councilman, Mallory, and Pearce,* in the study of 220 fatal cases of diphtheria, found bronchitis in most cases. The mucous membrane of the larger bronchi was reddened and covered with exudation, and usually small drops of pus could be forced from the smaller bronchi on pressing the cut surface of the lung. In 42 cases there was a fibrinous exudation in the bronchi, forming in the larger bronchi a distinct membrane, and completely filling the smaller.—Ed.]

5. **Bronchitis Due to Nervous Disturbances.**—These forms are very obscure and hypothetical. As it is certain, however, that the circulation of the bronchial apparatus is under nervous control, and that diseases of the brain and of the vagi have been followed by catarrh of the lungs which in its sudden and peculiar course differs from all our usual observations, I believe that this subdivision should be recognized, following the example of Marfan, who distinguishes *bronchitis of an angioneurotic* type and an *asthmatic* form. It is always doubtful how far the nervous element plays a part in these cases, and whether it is not rather the circulatory disturbance that occasionally leads secondarily to bronchitis. To describe a special asthmatic form of bronchitis is in many respects very convenient; for when, as is not at all infrequent, the symptoms of bronchitis are accompanied by asthmatic attacks, or attacks of dyspnea in which there is an inspiratory spasm and the expiration is reduced to a minimum, the bronchitis, because of these symptoms, presents a special type. In many cases the disease really begins as asthma, and it would therefore seem more correct to describe this form under the head of asthma, as heretofore; but in other cases such a classification is out of the question. Violent fits of coughing with intense dyspnea occur in all possible forms of bronchitis. Simple dry catarrh often gives rise to attacks that closely simulate asthma; and all the other forms, especially chronic catarrh, may have the same effect. In the capillary bronchitis of children the presence of this asthmatic element may lead to mistakes in diagnosis, such as whooping-cough, for instance. There are even individuals who appear to have a tendency to the occurrence of paroxysms, either congenital or the result of changes in the circulatory apparatus; we may say that an asthmatic element mingles with the bronchitis, although we cannot speak of a genuine asthma. In such cases the term asthmatic bronchitis is justifiable. I believe, however, that the introduction of this term is attended by the danger of great confusion; even now much mischief is sometimes done by its slovenly use. The best opportunity is given by this means to neglect a thorough investigation and observation, to throw

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* *Jour. Boston Soc. Med. Sci.*, vol. v, No. 5, 1900.

all such cases under the heading "bronchitis," and to treat them schematically. If the bronchitis assumes a special character, there must be a special reason for it. Of course, if the reason cannot be found it is most convenient to say: "Why, all this may occur in an ordinary case of simple bronchitis." Believe it who will; pray, what do you mean by "*simple bronchitis*"?

6. **Bronchitis in Constitutional Diseases.**—The causal relationship must be thoroughly proved before being accepted. There seems to be a true gouty bronchitis, in which urates are excreted through the bronchi. The establishment of a form of bronchitis in arthritis, in neuroarthritis, in herpes, and in the lymphatic diathesis, of which we read much in the French literature, is an attempt to subject to scientific investigation the difficult problem of the constitution, which must necessarily play its part in every pathologic condition. These attempts have so far resulted in nothing more substantial than words. Although it cannot be denied that the question has excited a lively interest and has been made the object of much ingenious speculation, we are unable to apprehend and prove scientifically the peculiar influence of individuality on the course of disease When a diabetic or a corpulent individual is attacked with bronchitis, it is not in itself a special kind of bronchitis, although it may in diabetics increase the tendency to tuberculosis, and in the corpulent favor development of cardiac disease.

7. **Bronchitis in Infectious Diseases.**—After the communication of Gintrac * we can no longer doubt the occurrence of bronchitis in intermittent fever. This author observed a form of bronchitis that comes on daily, runs its course with chill, fever, and sweat, and is quickly cured by quinin. The bronchitis of typhoid (enterica) is well known; it is so constant that we can regard it as one of the chief symptoms of the disease. It has its chief seat in the smaller bronchi, and is characterized by the fact that, in spite of the great extent of the disease, the cough and discharge are reduced to a minimum and are often entirely absent, as if the normal reflexes were abolished by the pyrexia. Under the influence of a cold shower-bath we often see how the patient begins to cough, and how the discharge also is increased. The bronchitis of other infectious diseases cited in the classification needs no further elaboration.

8. The **intoxications** which give rise to bronchitis are not numerous. Those which work through poisonous gases have already been mentioned, but their effect is especially on the mucous membrane. Here we have to do more particularly with those injuries which continue to act after the poison has reached the blood or the stomach. Thus we occasionally speak of a bronchitis in ammonia poisoning, when the poison was not inhaled, but swallowed. It may well be asked, however, whether in these cases the corrosion of the throat and epiglottis, which is always present, interferes with deglutition,

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* "Dictionnaire de Méd. et Chir. pratiques," Article on "Bronches," p. 569. He cites especially a description of Bouchard, *Journ. méd. de Bruxelles*, 1857.

and causes the saliva and other products to run into the trachea, must not be blamed as the direct cause.

A bronchitis has also been observed following the use of bromin and iodin, in carbolic acid poisoning, and in poisoning with cantharides. In the first three, it may be regarded as certain that a small portion of the poison reaches the bronchi through the blood. In the tracheobronchitis which is due to cantharides poisoning the mechanism is not so clear. It is certain that a very peculiar irritaion of the respiratory tract takes place.

By far the most frequent, and one of the most important, forms of bronchitis is that which is due to *alcoholic abuse.* It is one of the most wide-spread forms of chronic bronchitis, and is especially characterized by a tendency to emphysema and the formation of cylindric dilatations of the bronchi. A portion of the alcohol is excreted through the bronchial mucous membranes, and in this way exerts its injurious influence on the musculature, on the blood-vessels, and on the epithelium. This explains the flaccidity and hyperemia of the bronchi; the epithelium loses its cilia; masses of mucus accumulate; and it is easy to understand the development of the highest degree of emphysema, when we consider how persistently people expose themselves to these influences. Now, these catarrhs are, as a rule, erroneously ascribed to cold. All these broncho-catarrhs with emphysema, which we find so frequently in workmen, are drinkers'-catarrhs (Schnapps-katarrhe).

Drinkers'-catarrh is the most perfect example of an exhalation catarrh. Iodin catarrh belongs to the same category. Another example is furnished by the proof that in carbolic acid poisoning a severe tracheobronchitis may occur which was formerly explained by the fact that a portion of the carbolic acid, instead of being swallowed, found its way into the trachea. But that this view was not sufficient to explain the conditions has been very decisively shown by the experiments of Wachholz.* Finally, it is not improbable that in certain pathologic conditions the body itself elaborates injurious products, which are excreted by the mucous membrane of the bronchi and excite a condition of irritation and inflammation.

9. **Bronchitis in Diseases of the Upper Air-passages, of the Lungs, of the Pleuræ, of the Mediastinum, and of the Sternum (Kyphosis and Scoliosis).**—These are to be explained in part by extension, but still more by the injurious influences on the act of respiration, which are unavoidable. They often favor the penetration of dust, and then affect the nasal breathing. Diseases which bring about a diminution in the size of the thorax have as their prototype bronchitis due to kyphosis and scoliosis. Here the lung is too small and the movement of the thorax is too limited, so that the power of aspiration is impaired. As a consequence of this, the circulation is impeded, especially in the lower portions of the lungs [a passive congestion is established]. All forms of intercostal neuralgia are connected with these difficulties,

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* *Deutsche med. Wochenschr.,* 1895, Nr. 9.

and interfere with the breathing. Hence in the worst cases the same condition develops as is found in mitral bronchitis.

10. **Bronchitis in heart-disease** deserves especial mention. The cases have been divided into those of mitral and those of aortic insufficiency; there are, however, also mixed and other types. The first-named form, mitral bronchitis, is the one usually seen. Lasègue says it is characterized by hypostatic congestion of the lungs, with small, moist râles, diminishing from below upward. A small amount of fluid is usually present at the base. He declares that the dyspnea which is present is especially characteristic, occurring after the slightest exertion, while cough and expectoration are almost unnoticeable; the pseudo-asthmatic attacks that are seen in emphysematous patients, occurring at night without any apparent reason, are entirely lacking. The sputum is mucopurulent and characterized by the presence of the so-called *cardiac insufficiency cells*, which have given rise to all manner of discussion in recent times. For these large cells, which in healthy individuals contain black pigment, in these patients present red, rust-colored pigment which contains iron, is evidently derived from the blood, and has received the name "hemosiderin" (therefore "hemosiderin cells," the latest name).* The cells unquestionably originate in the alveoli of the lungs; this knowledge is chiefly based on the fact that they are very similar to the cells which are normally present and are generally regarded as desquamated epithelial cells. The name cardiac insufficiency cells is justified because they are found almost constantly, and in such numbers as to be considered characteristic, in the hypostatic conditions of heart disease and in no other conditions. It is stated that they are occasionally found in tuberculosis, pneumonia, and other diseases of the lungs, but this has no significance whatever. They are quite rare, I may say exceptional, findings, just as asthma spirals may be found in those who are not asthmatic, or tube-casts in the urine of those who have not Bright's disease. In fact, we generally find that certain symptoms are quite characteristic for one disease, but occasionally occur in many others. Aortic bronchitis, on the other hand, and the form occurring in arteriosclerosis, is not a characteristic, but an artificial form; although, according to Lasègue, it is associated with the presence of small scattered foci with small moist râles, and the patients have violent and convulsive attacks of coughing. As I have said before, I do not believe that these symptoms are due to bronchitis at all. Pure aortic insufficiency never leads to bronchitis, although stenosis does in its later stages, the bronchitis being quite similar to the form observed in mitral disease.

Myocarditis has also been credited with a special form of bronchitis. Huchard † calls this form *bronchite à répétition de la myo-*

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* Note here also the mania for nomenclature. As hemosiderin is far from being accurately known, the new name is no better than the old; however, it sounds so beautiful that I can promise it a certain lease of life.

† "Traité des maladies du cœur."

cardite scléreuse. It consists of ordinary attacks of bronchitis, which do not last long, but gradually become chronic and lead to a condition like that in mitral bronchitis, with severe asthmatic attacks.

The important thing is to know the fundamental type of mitral bronchitis and to remember that a whole group of cardiac affections, especially diseases of the myocardium, of the coronary vessels, and also of the aorta, may cause pseudo-asthmatic and stheno-cardiac conditions.

We have frequently mixed forms, but the fundamental form is always true congestive catarrh (*Stauungskatarrh*). As the bronchial veins empty through the vena azygos into the vena cava, the bronchi do not at first come under the influence of the venous engorgement in mitral and aortic lesions, and are not affected by these to any high degree until the engorgement extends backward through the right side of the heart. This is evidently the reason that in many mitral lesions we see a good compensation, without bronchitis, preserved for some time, for the anastomoses between the arteriæ pulmonales and the venæ bronchiales, which are sparingly present, become enlarged and so for a time prevent a burdening of the alveoli.

11. **Bronchitis in diseases of the digestive tract** s only an exceptional occurrence. Bouchard and Le Gendre claim to have observed bronchial catarrh as a frequent symptom in dilatation of the stomach; it might be of reflex origin or due to autointoxication. A distended stomach might also exert pressure on the posterior lower portion of the lung through the diaphragm. What I have observed of dilatation of the stomach, however, by no means corroborates this view.

The *bronchitis of dentition* in children may also be mentioned here. While many children at the time of dentition suffer from slight diarrhea, there are also some who remain constipated, and a tendency to bronchitis then manifests itself; whether there is really any deeper relation I dare not assert. It is certain that children at this time are susceptible, or, as skilled nurses say, are liable to attacks (*anfällig*). They are also more apt to develop bronchitis than in times when they are well. But I cannot regard a dentition bronchitis as a special form. I can make nothing of Gintrac's statement that "it is due to organic disposition peculiar to each child, in consequence of which the sympathetic influence of dentition manifests itself in one case in the follicular apparatus of the bronchi; in another, in the follicular apparatus of the intestine."

Marvelous descriptions of bronchitis from worms are found in some of the older books. It is true that, in individuals who are predisposed, cough may be excited by any form of peripheral irritation. I admit the possibility of a stomach, liver, intestine, spleen, or uterus cough; I would not even reject a kidney cough if a good clinical history was presented to me from a reliable quarter. But from reflex cough to bronchitis, it will be admitted, is a very long step. This worm-cough, then, is described as a dry, sonorous, extremely violent cough, occurring day and night, often every five minutes, in which some râles are heard in the chest, and which disappears when the worms are exterminated.

I also have my doubts about the assertion that bronchitis occurring in children with digestive disturbances, especially diarrhea, is a consequence of the intestinal catarrh. It is true that this coincidence is not infrequent in sickly children, but the one is not the consequence of the other. In conditions of malnutrition the bronchi easily succumb to slight injuries to which children are so frequently exposed. In many cases scrofula is no doubt the predisposing factor both for the intestinal canal and for the bronchi; and whether one or the other system, or both, are affected depends on the distribution of the scrofulous lesions, particularly in the glands.

12. **Bronchitis due to nephritis** is quite frequent, and we accordingly speak of albuminuric bronchitis. It is supposed that this form is due to uremic disturbances, but the impairment of the heart action, which is peculiar to nephritis, is no doubt partly responsible for its production. However, this form of bronchitis occurs commonly enough in acute nephritis, when there are no signs of heart trouble or uremia. The causal relationship must therefore remain undecided for the present.

Lasègue, who has made a special study of albuminuric bronchitis, distinguished three forms: (1) Bronchopulmonary edema of short duration (*oedème broncho-pulmonaire fugace et migrateur*), characterized by severe dyspnea coming on in paroxysms, and by the presence of crepitant râles in circumscribed areas of the lungs. (2) True albuminuric bronchitis, like the foregoing, characterized by dyspnea and cough, with occasional severe paroxysms, but presenting over a wide area, including all the lower portion of the lungs. (3) A form of very great severity, in which to the symptoms of the preceding forms are added areas of dulness and febrile movements.

[Johannes Seitz * reports two cases of acute bronchitis in children, which were followed by acute nephritis. Scarlet fever and other causes were excluded. A few days after exposure and consequent acute bronchitis, albumin in large quantity, blood, renal epithelium, and casts were found in the urine, which had previously been normal. Virulent Staphylococcus pyogenes albus and aureus and Streptococcus pyogenes were found in both urine and sputum. In one case albumin persisted for some time. The author considers both cases of nephritis due to transference of the bacteria—which had gained entrance through the bronchi—to the kidneys. This was the only report of cases found after an exhaustive review of the literature on the subject. The possibility and the probability of such relation are urged by writers † on the subject, but no cases are cited.—Ed.]

13. **Diseases of the skin** by no means predispose to bronchitis, and for the present I must regard the coincidence of the two as purely accidental. If the onset of a bronchitis is followed by improvement

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* *Correspondenz-Blatt für schweizer Aerzte*, XXVIII, 1898, 673.

† Lasègue, "Etudes Médicales," Paris, 1884.

in an eczema which was present, it is no proof that there is a direct relation between the two conditions. It is often observed that skin eruptions abate on the appearance of severe internal diseases, and Hebra has already sufficiently criticized the theory that eczema may give rise to metastasis in the internal organs. The only fact that remains is that any acute exanthema may involve the mucous membranes of the respiratory organs (enanthema).

[**Eosinophilic bronchitis** is described by Teichmüller.* The exact nature of the disease and its causes are not known, but it seems to be related to tuberculosis and also to a general neurotic family history. It is not uncommonly associated with rheumatism and is likely to follow exposure. It commonly runs a subacute or a chronic course. Among the symptoms are dyspnea; frequent cough; mucoid expectoration containing a large number of *eosinophile cells;* some pain in the chest; disturbance of digestion. Fever is usually not present. It is often difficult to distinguish the disease from tuberculosis, but the prognosis is good. The *treatment* recommended is hygienic and dietetic, giving potassium iodid if syphilis is suspected.—See also the article on Asthma for a discussion of this form of bronchitis. On the other hand, Fuchs † finds eosinophiles in the sputum in increased numbers in all affections of the respiratory tract, but especially in the fevers. Teichmüller's view, that in the early stages of tuberculosis the presence of increased numbers of eosinophiles in the sputum is a sign of good resistance, and therefore a point on which to base the prognosis, is considered by Fuchs too broad for practical purposes.—Ed.]

PATHOLOGIC ANATOMY.

Catarrh of the bronchi begins with a **hyperemia** of the mucous membrane. This is the consequence of, and the natural reaction to, the injury received. At first the hyperemia consists only in well-marked injection of many small blood-vessels, which stand out quite distinctly; soon, however, the redness becomes quite diffuse and small *hemorrhages* generally occur. In a certain number of cases these quickly disappear, but in the majority of cases they last for a variable time.

These hemorrhages either last as long as the catarrh, or they gradually disappear, in spite of the continuance of the catarrh, and give place to an anemic condition. The principles which govern this occurrence are quite unknown.

In a certain number of cases the hyperemia precedes the catarrh, and then acts as a predisposing factor, especially in the so-called *engorgement hyperemia.* Of the hyperemia it is to be observed that it may be entirely lacking at the autopsy, although it was present to a very high degree during life. This is especially observed in the bronchi, on account of the great number of elastic fibers contained in their walls.

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* *Deut. Arch. f. klin. Med.*, Aug. 18, 1899.

† *Deut. Arch. f. klin. Med.*, vol. 63, p. 427, 1901.

A more abundant serous **transudation** now appears, which is regarded as the result of beginning alteration of the epithelial wall. The nature of the transudate varies greatly according to the cause of the inflammation: it is now richer, and now poorer in albumin. At times red blood-corpuscles may appear in it in great numbers, while at other times they are almost entirely lacking. The same is true with regard to the wandering out of the white blood-corpuscles. The transudation is not merely the expression of a filtration process, but involves many complicated relations, which have not been made the subject of investigation.

The transudate produces **hypertrophy** of the mucous membrane, which appears very rich in fluid and assumes a peculiar velvety character. In the smaller bronchi this swelling may obviously lead to such a *narrowing* of the lumen that the passage of the air is attended by great difficulty, and finally becomes quite impossible. This narrowing naturally develops earliest in young children, and we accordingly see that in these subjects catarrh of the finer bronchi must in itself represent a disease that is dangerous to life.

The longer the catarrhal inflammation lasts, the more obstinate is the swelling, and the same obstinacy is noticed in the frequent recurrences. In the more chronic conditions it therefore leads to proliferation in the glands, while the interglandular tissue is edematous and shows many nuclei; in this way a true hypertrophy of the mucous membrane may take place. This hypertrophy of the mucosa is well marked in certain old catarrhal conditions.

The **glands** appear very prominent on the surface of the mucous membrane. From the mouth of a gland duct a small drop of fluid can be expressed consisting of mucus which is turbid from the presence of desquamated cells. The orifices are much enlarged, and may even be distinctly visible to the naked eye. They may even be mistaken for submiliary ulcerations.

In addition to the glandular changes, there is always an abundant new formation of **blood-vessels,** which, under certain circumstances, form villous growths, cause bulging of the mucous membrane, and alter the surface after the manner of papillary formations. These new blood-vessels also penetrate the muscular coat and even the cartilage, which may soften and break down in places, to the serious detriment of the bronchial wall.

In marked contrast to these cases, there are others in which no such hypertrophy occurs, but in which the new growth of connective tissue leads to cicatricial contractions: the glands and blood-vessels are compressed and an **atrophic mucous membrane** results. In many such cases it looks as if the atrophic form were the real terminal condition, the hypertrophic form representing an earlier stage. But in opposition to this view, a similar hypertrophy has been found in well-authenticated old and chronic cases, so that we are forced to believe that the hypertrophic process does not necessarily end in atrophy. In this region also, as in the large glands of the abdomen, it is not yet decided what factors determine whether the one or the other process shall develop. One would think that these questions could be most easily answered in the case of the pharynx and larynx, which are easily and constantly accessible to observation. Here also hypertrophic and atrophic forms of catarrh have been observed, but here also clinical and pathologic observations have failed to yield harmonious results, so that one would explain the other.

A few points have, however, been established; they are:

1. The tendency to *hypertrophic* catarrh manifests itself especially in mucous membranes that contain many glands, as the participation of the glands is necessary to the development of these chronic hypertrophies; in structures consisting only of epithelium and connective tissue catarrh leads to an *atrophic* condition. On this depends the fact, which at present interests us especially, that the further the chronic catarrh extends toward the finer bronchi, the more it assumes an atrophic character. It is possible, however, for the hypertrophic form to develop even in the terminal ramifications. To be sure, there are no longer any open glands, but the mucous membrane still contains follicular tissue.

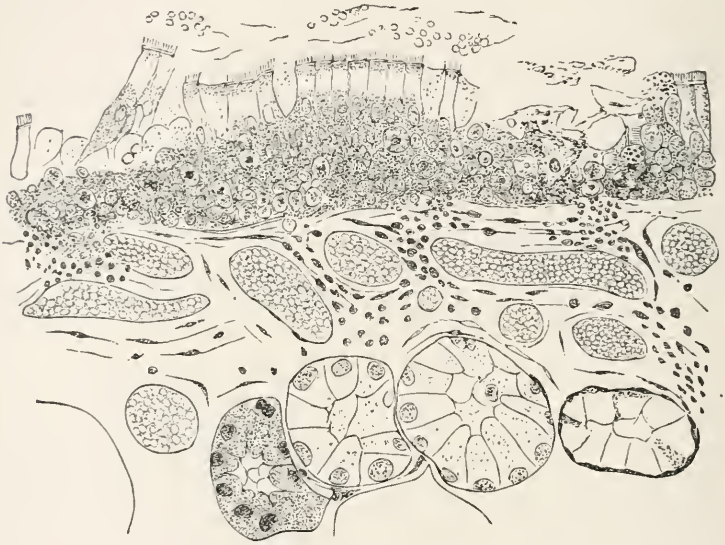

FIG. 4.—Acute catarrhal bronchitis.

2. In healthy individuals there is no *à priori* tendency to the development of an atrophic catarrh in mucous membranes that are well supplied with glands. If the catarrh assumes such a character,

the question at once suggests itself, Has not the catarrh developed on an abnormal soil? An abnormal soil of this kind is prepared chiefly by syphilis and alcoholism. Certain injurious local influences, as tobacco smoke and dust-inhalation especially, provoke a hypertrophic catarrh.

Secondary Changes.—In consequence of the changes in the mucous membranes, the epithelium is still further loosened; the swelling and serous infiltration naturally exert their full effect whether an injury has already destroyed the epithelial elements or the loosening now occurs for the first time. The cylindric epithelial cells become swollen, part of their cilia are lost, the nuclei become more distinct, and the cells are cast off and enter the secretion. Others lose their uppermost, cilia-bearing portion, become goblet-shaped, and pour their contents out on the free surface. Some of the epithelial cells thus become mucous cells and others become goblet-cells. When the inflammation becomes still more severe, all the epithelial cells are cast off, the uppermost layer is formed of round cells, the surface assumes the characteristic smooth and shiny aspect due to the basement membrane being laid bare (Ewart and other authorities), loses its smoothness entirely, and becomes rough.

In cases in which the inflammation lasts a long time and in which there is a tendency to recurrence or to frequent exacerbations, the layer of smooth muscle-fibers lying in the submucosa is more or less changed and the muscularis itself becomes infiltrated. The white blood-corpuscles which have wandered out settle around the smooth muscle, and if this condition lasts for some time, atrophy of the muscularis necessarily results. It may even give rise to bronchiectasis.

Finally even the cartilages no longer offer resistance; they are also attacked, and an early ossification follows. In older people there already exists a tendency to calcification, and when this is united with the ossification, the bronchi may ultimately be converted into perfectly rigid tubes.

In most cases, then, chronic catarrh is an atrophic catarrh; there is, however, also a chronic hypertrophic catarrh. The mucous membrane does not lose its velvety appearance, which it had in the acute inflammatory condition, but rather shows larger elevations, while the vascular growths increase and bring about a marked constriction of the lumen of the smaller bronchi. Some authors have even spoken of an *obliterating bronchitis.* But such cases must be quite isolated and rare.

A natural limit is set to the deeper penetration of the inflammatory changes by the thick layer of elastic fibers which is situated under the submucosa. These, however, naturally become thinner and thinner the further they are from the main trunk, hence the catarrhal inflammatory changes traverse the walls much more quickly in the smaller bronchi than in the larger ones. But if the whole wall is attacked, it becomes infiltrated with an inflammatory exudate, and the peribronchial connective tissue becomes thickened. In the secondary and tertiary bronchi, therefore, chronic catarrh quite usually leads to *peribronchitis*, while this phenomenon is rare in the larger bronchi. Peritrachitis, however, is not an unheard-of event.

Rindfleisch in his time described quite minutely how a true **ulceration** of the mucous membrane may be added to the catarrh. This ulcerative process develops from the mucous glands. On pressure there exude from their orifices small amounts of tenacious mucus which appear like gray translucent pearls. If—so this author says—there appears in these masses a narrow yellowish-white seam, it is a sign that the ulcerative process is beginning; for this is the pus furnished by the subepithelial connective tissue of the affected gland ducts. Gradually a flat ulcer develops from the mouth of the gland duct; extension to the gland itself soon follows, and the destructive process then attacks the submucous connective tissue and may even reach the perichondrium.

It is undoubtedly possible that in a simple catarrh such complications may occur, but they do not belong to the disease at present under discussion; they represent merely complications, and we should think, in the first place, of a tuberculous or syphilitic infection. It is certain that many kinds of microbes may cause the destructive process, but these forms also represent complications which need closer investigation and which do not belong to the simple catarrhal types. True catarrh never leads to such destruction; this I must insist upon as a fundamental characteristic.

On the other hand, simple catarrh leads to a certain swelling of the follicular apparatus; to a genuine *adenitis* without any tendency to pus formation; to inflammation of the lung tissue, so-called *catarrhal pneumonia;* to *bronchiectasis;* to *emphysema* of the lungs; to disturbances in the pulmonary circulation; and, finally, to *fatty heart* and *dilatation* with all its consequences, which are explained on the basis of insufficient oxygenation of the red blood-cells.

[**Atheroma** is frequently observed in the middle-sized pulmonary arteries in the subjects of chronic bronchitis. A separate form is characterized, according to Virchow, by a *peribronchitis fibrosa chronica*, the fibrosis in some cases extending along the lymphatics to the entire interlobular stroma. Instead of emphysema, the lung then presents diffuse condensing fibrotic changes.—Ed.]

GENERAL SYMPTOMATOLOGY OF THE VARIOUS FORMS OF BRONCHITIS.

On inspection, we see the **respiration** changed only in severe cases of bronchitis in children and old people, while the strong man in middle life shows no disturbances of the respiratory rate; such difficulties at this age indicate a further complication. In children, however, the obstruction soon becomes so great that, with any extension of the catarrh, dyspnea develops; at times, when the catarrh has attacked the smaller bronchi, the dyspnea is such a prominent symptom that one is inclined to diagnose the case as pneumonia. It may be added that febrile movements, which are most marked and earliest in children, always cause an increase of the respiratory rate.* With these, we see also, especially in capillary bronchitis, that inspiratory *retraction* of the lower ribs and of the epigastrium which is so much more pronounced in acute constrictions of the larynx: the lungs are unable completely to follow the lowered diaphragm and the ribs are drawn in to fill out the space in which negative pressure exists.

After different predecessors, among whom Biermer and Gerhardt are the most distinguished, Riegel † subjected the **dyspnea** to a very careful study. He distinguishes an *expiratory*, an *inspiratory*, and a *mixed* form. The latter consists of a simple increase in the rate of respiration, in which inspiration and expiration are equally affected; it is always found in simple acute bronchitis. Only in exceptional cases do we find any other type of breathing in this disease. The expiratory dyspnea occurs in extensive chronic bronchitis with a small amount of viscous secretion. On theoretic grounds we should expect a general slowing of the respiration with expiratory dyspnea, but the authors have not observed this; they have observed either mixed dyspnea (simple hastening of the respiratory movements), or expiratory dyspnea, sometimes with and sometimes without increase in the rate. The latter belongs, according to Riegel, to the more severe and protracted forms.

Lebert speaks only of the increase in the respiratory rate. Sée says that two distinct causes occasion the dyspnea—the intensity of the catarrh and the height of the fever. The second governs the disturbances to the extent that, when the fever is lacking, there is very little disturbance of respiration, even in cases of very widespread bronchitis. When dyspnea occurs, as in children, nervous individuals, young girls, and old men, it is chiefly of the expiratory type.

In many cases the *expansion of the thorax* is greatly altered; while certain portions can expand only a little on account of the obstruction of the bronchi, others expand all the more, and it is, accordingly, stated by Riegel that in men with chronic catarrh of the lower lobes a truly feminine type of respiration develops.

Finally, there are cases with distinct irregularity of respiration. This form of dyspnea, however, is to be ascribed not to simple catarrh, but to a certain nervous disposition. It is often observed in hysteric and neurasthenic patients, especially when they are thinking of their breathing. The cases that sometimes occur in children in severe stages of bronchopneumonia do not belong under this heading.

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* Ackermann, *Deutsches Archiv für klin. Medicin*, vol. II, p. 361.

† *Deutsches Archiv für klin. Medicin*, x.

In estimating the frequency of respiration, it is necessary to remember that the healthy adult man makes 14 to 16, and the woman 20, respiratory movements per minute. In children the number is always higher; for instance, in the new-born 30 to 50, during the first years of life 25 to 35. In sleep the number is always reduced.

The **percussion note** is not changed; but in catarrhs which have lasted for some time the borders of the lungs which cover the heart and fill the complementary spaces are distended, so that the resonant area obtained may be very similar to that observed in acute emphysema. This condition is often called *acute emphysema.*

On **auscultation,** a great variety of râles may be heard. Although it must be admitted that there are cases in which râles are entirely lacking, this absence is only transitory. In other words, by coughing and breathing the râles may be made to disappear, but only to return after a short time. Even so-called dry bronchitis is always accompanied by râles. The râles of bronchitis are recognized by the fact that they have neither a crepitant nor a ringing character [nonconsonating]. It is a matter of practice; they can be learned, but they cannot be described. Some of the râles are caused by the mucus which is in the air-passages; it is driven hither and thither by the air-current, and the air is driven through it in larger and smaller bubbles. The bubbles reach a variable size, depending on the diameters of the tubes. And so we distinguish large, medium, and small bubbling râles. It is also clear that in the smallest branches the bubbles become proportionately less; hence the character of the râles permits us to form an opinion as to the kind of bronchi in which they originated. These râles are called moist râles. Small moist râles are sometimes included amongst crepitant râles, but this classification is incorrect, since crepitant râles are not produced in the bronchi at all. I cannot doubt that they have their seat in the infundibula. The most inexperienced clinician can distinguish them from moist râles, even from the smallest ones, by the fact that they are purely inspiratory. (Exception, Penzoldt, "Deutsche med. Wochenschr.," 1877.)

The view that râles indicate the presence of fluid in the bronchi, through which the air is forced in bubbles, has been attacked by Traube. He explains the occurrence of râles by the theory that the viscous bronchial contents are torn loose as a whole from the bronchial wall during both inspiration and expiration, and that the air rushes in to occupy the spaces made vacant by that process. Traube's reasons for this view are based on our knowledge of crepitant râles. Wintrich has already shown that true crepitation, as described by Laennec, is produced by the infiltration of sound lungs, and Traube calls attention to the fact that in the parenchyma of every normal lung, even in that of an animal that has just died, the most perfect crepitation can be produced by careful, rhythmic pressure with the stethoscope placed against the pleura. I must, however, protest vigorously against confusing small bubbling (moist) râles with crepitant râles. The *fine* bubbling (vesicular) râles of our authors are often the same as crepitant râles; but some use the term *fine* bubbling (vesicular) râles in the sense of *small* bubbling (moist) râles.* It is therefore better not to use the term "fine bubbling râles." There is such a thing as a small bubbling râle; it belongs to the same class as medium-sized bubbling râles, is a sign of mucus, fluid, and air in the bronchi, and is therefore a symptom of bronchitis. There is also such a thing as a crepitant râle or crepitation. It is a distinctly inspiratory phenomenon; it is the sound which, as Wintrich has shown, is present in the healthy lung, and which, as Traube correctly stated, is heard so well when there is a small residue of pleuritic exudate. It is caused by the entrance of air into collapsed infundibula and alveoli, and is strongest and most lasting when some secretion glues the walls of the alveoli together after each expiration; otherwise it is heard only when the first deep breath is drawn, and then disappears. It is not a sign of bronchitis, although it occurs in bronchitis; and it is not a moist râle, but quite a peculiar sound. Hence the term "crepitation" or "crepitant râle" is to be preferred, as it does not lead to misunderstanding.

Excellent as this sharp differentiation may be in theory, it must be confessed that in practice there are cases in which crepitation and small bubbling râles merge into one another so imperceptibly that to insist on one or the other expression for the single sound that is heard is purely arbitrary. Since it is impossible to draw the line accurately between alveoli and bronchi, it is not to be wondered that crepitation is occasionally heard in bronchitis.

In contrast to the moist we have dry râles, which are quite as characteristic of bronchitis as are the former. Their production depends not on the presence of fluid, but on vibration of the bronchial walls. The sound is due chiefly to the irregular swelling of the mucous membrane, so that the entire wall of the bronchus is thrown into vibration by the current of air as it forces its way through the narrow places. Thus are produced the sonorous and sibilant ("whistling" and "purring") râles. When they reach a certain intensity, these dry râles are felt by the palpating hand, and give the sensation of a cat's purring (*frèmissement*, fremitus, or thrill).

The sonorous râle (*ronchus sonorus*) is produced in the larger bronchi; the sibilant râle (*ronchus sibilans*) in the smaller. When the whistling element is less clear, the term sibilant râle in its strict sense is no longer appropriate, and if we wish to burden the terminology still further, we may use the term "sighing respiration" (*Giemen*).

Changes in the Respiratory Murmur.—There is a change in the respiratory murmur itself, in so far that it may assume a very rough and sharp character, and with it is connected a lengthening of the expiratory murmur. This lengthening shows that the air meets with hindrances to its exit in the air-passages; it is therefore a very frequent phenomenon, which occurs in all infiltrations, in the presence of growths, and in all processes that cause compression. The greatest caution is necessary in drawing conclusions from a symptom that may mean so many different things.

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* As, for instance, Riegel, in Ziemssen's "Handbuch," vol. IV, H. 21, p. 114.

As there is an accentuation, so there may also be a weakening of the respiratory murmur. This is due to the superficial character of the breathing, especially in those cases in which it is attended by pain. But mucous obstructions and, in general, any kind of narrowing and obstruction in a bronchus may also cause weakening of the respiratory sounds in the region supplied by that bronchus. If such a hindrance is removed by a violent fit of coughing, loud or even accentuated breathing is heard for a time where before nothing at all was audible.

Cough is a convulsive expiration accompanied by a loud sound. This sound is occasioned by contraction of the muscles that close the glottis. In coughing a simultaneous action of the expiratory muscles and the muscles of the glottis takes place. The object of cough is to expel from the bronchi any foreign bodies, including mucus and pus when they act as foreign bodies. How effective the cough is for this purpose every one can observe for himself.

Cough may be produced intentionally, but is usually a reflex act, which originates from various regions of the body, differing in susceptibility according to the individual's disposition. Among structures that are without a doubt normally endowed with such a sensitiveness may be mentioned the larynx, including the upper portion of the trachea, the mucous membrane at the point of division of the trachea, and the mucous membranes of all the smaller bronchi. The reflex is produced most energetically at the point of division of the trachea and in the subglottidean and interarytenoidean mucous membranes. The nerves that carry the impulse centripetally are the superior and inferior laryngeal and the pulmonary branches of the vagus. In fact, after separation of these nerves from the region supplied by them, cough can no longer be produced. In coughing, as in all reflex acts, *irradiation* plays an important part. This fact should never be lost sight of. We find that in especially predisposed individuals cough is occasionally due to irritation in portions of the body far removed from those just mentioned. In the great majority of these cases the sensitive region lies within the realm of vagus influence; hence cough may arise from irritation of the posterior nares of the pharynx,* of the external auditory meatus, of the whole tracheal tract, of the pleuræ, of the esophagus, of the stomach, of the spleen and liver, and, finally, of the intestine.

The question whether irritation of the lung itself produces cough has been much discussed; all authors are now of the opinion that cough is caused by irritation not of the lungs, but of the bronchi.

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* Ruault, "Toux Amygdalienne," *Annales de laryngologie*, vol. 1, p. 154, 1887-1888.

There are also communications on the subject of cough arising from irritation of areas which certainly are not under the influence of the vagi; thus, we have *nasal*, *dental*, and *uterine* cough. Kohts has caused cough experimentally in dogs by stimulation of the medulla oblongata; but cough in diseases of the brain is at the present time not known to us. In hysteric patients any peripheral irritation may cause cough; dental and uterine cough are hysteric forms.

The manner in which the cough really acts is evident. Its object—to force mucus, etc., upward from the bronchi—is usually accomplished by the ciliated epithelial cells; but when these are not sufficiently active to bring about the desired result, their efforts are aided by the act of coughing. The pressure in the interior of the bronchial system is greatly increased; this is suddenly followed by a diminution of pressure, and the air rushes toward the exit. The sudden and forcible rush of air naturally carries small objects along with it. There is some danger of their being drawn in again with the inspiration, as the cough is followed by an inspiration of greater force than usual; but the force of the inspiration always remains far behind that of the expiration, especially at times when, as in cough, the closure of the larynx has still further increased the pressure in the bronchial tree.

That this reflex may at times be very distressing without apparently subserving any good purpose is well known. Cough, like all reflexes, is very valuable, but in individual cases it may be very injurious.

We usually distinguish a *moist* and a *dry* cough; we speak also of *irritative* cough and of *convulsive* cough. Irritative cough is usually a dry form in which the irritation is of special intensity and quite irresistible; an inconsiderable swelling of a circumscribed portion of the body, or a minimal foreign body (for instance, bits of food), may elicit an irritative cough if it comes in contact with the areas most sensitive to reflex stimulation.

The cough is ushered in by a peculiar feeling of tickling in the throat; if one takes the trouble, he can often suppress the cough by conquering this feeling of tickling. When the sensitiveness of the nerves is reduced to a certain degree, cough is no longer produced; somnolent patients in whom tracheotomy had been performed have had the trachea sounded, and even the most sensitive places touched, without being made to cough. Thus, in very weak individuals large amounts of mucus may collect in the bronchial tree and in the trachea without the slightest impulse to cough being excited. In this respect also the behavior of cough corresponds with that of the other reflex acts.

Very violent coughing leads occasionally to vomiting. When this happens in adults, we must always regard the matter as very serious; a marked disturbance of nutrition must lie at the bottom of it; an adult never vomits in a simple bronchitis. It is different with children, in whom any severe attack of coughing has this result; in the paroxysms of whooping-cough vomiting is an essential part of the clinical picture.

Sputum.—Next to cough, expectoration is the most important symptom of bronchitis; it is lacking only occasionally in rare cases of dry chronic catarrh. In small children, in very old people, and in very debilitated and apathetic individuals who generally swallow the sputum, it cannot, of course, be observed. Yet with some attention one can, if he is on hand at the moment of the spell of coughing, get some sputum, even from the smallest children. The sputum contains very few characteristic elements. Many times the mistake has been made of regarding and describing as important the normal secretions which are, of course, mingled with the pathologic discharge. (Compare page 33.)

As a very characteristic element of bronchitic sputum, I may mention the *ciliated cells.* They are often quite unchanged, of elongated form, the lower end showing long processes, while the upper end presents the characteristic, sharply contoured membrane, often bearing distinct cilia. These forms may be very much changed; the upper portion may degenerate and a portion of the contents be expelled, giving the appearance of the typical goblet-cell, which, however, is rarely seen.

Besides these forms, which are so characteristic and easily recognized, there are found *mucus* and *pus cells.* They are round and only exceptionally appear to contain pigment. I have myself observed no case in which the presence of the pigment was unquestionable. In their protoplasm we see only a certain granulation, but rarely a real granular degeneration. Eosinophile cells, which they otherwise resemble, but which are recognizable by a very beautiful and characteristic granulation, do not come in question here, although it is possible that at times one might wander in by chance. [See Varieties of Bronchitis, p. 118, and article on "Asthma."]

The presence of all kinds of squamous cells from the mouth, throat, and vocal cords is, of course, to be ascribed to lack of care in making the preparation. These cells cause no difficulty in making the diagnosis. Some ciliated cells from the nasal cavity may also be present, but are of no significance.

In the consideration of pathologic sputum we base our classification generally on the appearance. Biermer's "Arbeit" is still the standard on this subject.* He distinguishes: (1) tough, mucous, transparent sputum, poor in cells—*mucoid* sputum; (2) sputum containing many cells and much mucus—so-called *mucopurulent* sputum; (3) *purulent* sputum. These are the varieties observed in the ordinary forms of bronchitis. In certain cases, which are not to be discussed here under the head of simple bronchitis, we find seromucous, fetid, and fibrinous sputa.

Mucoid sputum belongs especially to the initial stage of acute bronchitis. It is translucent, stringy, and contains many air-bubbles.

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* Biermer, "Die Lehre vom Auswurf," Würzburg, 1855.

Microscopically it shows: (1) A few white and red blood-corpuscles. (2) Desquamated cells of bronchial epithelium, which attract attention on account of their graceful form and the ciliary border which is often well preserved (it may even show ciliary movement in fresh preparations). The occurrence of these cellular elements, which evidently indicates a decided pathologic condition, has until now been dismissed with a few general remarks. There are cases in which they occur in such large numbers that they give to the sputum a peculiar character (desquamative broncho-catarrh); but in the great majority of cases they are very few, and one must search through several fields of the microscope in order to find them. (3) Drops of myelin may be mingled with the last-named variety of sputum; but they are very few and quite isolated, and do not present the complicated forms found in other sputa. This sputum is called by older authors *sputum crudum.* It consists only of the abundant secretion of the mucous glands and the epithelial cells cast off from the surface. According to the abundance of the secretion, the sputum may be very viscous or more fluid. A scanty secretion may cling to the mouth with such tenacity that it must be wiped off with considerable force. When the secretion is very abundant, a sputum is produced which Biermer has denominated *seromucous sputum* The boundary-line between mucoid and seromucous sputum is, of course, extremely arbitrary.

Mucopurulent sputum is distinguished from the preceding kind chiefly by the large number of pus cells, which, as a rule, give to the discharge a more yellowish color; or at least yellowish flakes appear in the white masses. We can thus distinguish between the mucous and purulent portions in the sputum. The microscopic elements are the same as in the foregoing variety, except that here the pus cells are abundant and the cylindric cells few; they have often a peculiarly plump form and the ciliary border is injured or may be entirely lacking. Besides this, cells containing fat droplets are more abundant. Mucopurulent sputum occurs in simple chronic bronchitis.

Purulent sputum: The color is no longer whitish, and there is no pure white portion; it is yellowish, even greenish, and may be brownish, and in the air often becomes much more pronounced. Cylindric cells are rarely found; the morphologic elements consist mainly of pus cells which in acetic acid show their lobulated nuclei very distinctly; there are also some which show single round nuclei. There are many fat droplets in the different cells and also free in the sputum; they are mostly very minute. Frequently they form large clumps, and then give the impression of large cells that have undergone fatty degeneration; they keep well together, but are, as a rule, so entirely without any indication of nucleus or protoplasm that we cannot be certain whether they should be regarded as cells, although they may have developed from cells. The bright, glistening, quite sharply contoured, and dark droplets are entirely unaffected by alkalies and acids, and are very characteristic of chronic forms of bronchitis.

An attentive observer cannot fail to be impressed by the fact that there is a purulent catarrh with much fatty degeneration, fat drops, and balls of fat crystals, and another form of purulent catarrh in which we have to search long for any pathologic products. The reason for these differences is not clearly understood.

According to the appearance, Biermer has divided purulent sputa into three sub-classes; this division is entirely worthless. One of these classes is made up of the famous coin-shaped sputum, from the form of which one can determine nothing. [It will be necessary, for practical purposes of treatment and prognosis, to classify the sputum in accordance with the bacteriologic findings.—Ed.]

With regard to the **general physical properties of sputum,** it has also been said that the secretion which fills the finest bronchi is not mixed with air, and thus, being specifically heavier than water, sinks in it. If it possesses the necessary tenacity and cohesiveness, it is said to take the form of the smaller bronchi and to stick fast to that part of the sputum which, coming from the larger bronchi, is mixed with air and therefore floats on the water. Accordingly, when the sputum is poured into a vessel of water, it forms on the surface a mucoid layer with fine threads of hyaline material depending from it. This description is somewhat arbitrary; it has never been proved that these threads, which hang down in the water, and may be seen in any sputum that floats on the surface, originate from the finest bronchi; when the differences of caliber and arrangement are considered, it is not even probable. That in a heterogeneous mass like sputum of this kind is, the specifically heavier portions should sink to the bottom and bring down with them fine threads of mucus is quite conceivable. I would, therefore, reserve judgment on the above description until better reasons are brought forward in its support.

The discharge is, as a rule, examined only by its gross appearance; it is rarely brought to the microscope. The chemical examination has no practical value, and is used in only a few cases. Recently a chromo-chemical method of investigation has been proposed which puts us in a position to form more definite opinions than formerly concerning the albumin, mucin, and fibrin contents of the sputa. A piece from the size of a pea to that of a bean is placed in a test-tube; this is filled half full of a 0.5% alcoholic solution of sublimate and then shaken until the clump of sputum is broken up into the finest fibrils (about five minutes). After these have settled, the alcohol is poured off and the test-tube filled two-thirds full of distilled water. Three drops of the coloring mixture, presently to be described, are then added and the flakes carefully stirred for from three to six minutes, after which the solution is poured off. Albumin, protoplasm, fibrin, and connective tissue are colored reddish, and mucin a greenish color. The coloring fluid consists of one part of Grübler's Biondi tri-color mixture to thirty parts of water. This solution may be kept about four weeks. The mixture must be perfectly neutral. The least excess of stain changes the brownish-red color to a Bordeaux-red, while an alkali changes the color to a greenish shade. (S. Schmidt, followed by Zenoni.*)

Sputum has occasionally been examined chemically. Bamberger † gives such an analysis of the sputum in chronic bronchitis. It contained of water, 95.622 parts; of organic substances, 3.705 parts; and of inorganic salts, 0.673 parts. Among the latter, chlorin and sodium stand at the head, then follow phosphoric acid and potassium, while all the others are present in minimal amounts. The combination differs from that of pus essentially in the fact that the latter contains decidedly more organic constituents, while among the inorganic, the phosphoric acid approaches the chlorin in quantity, if it does not exceed it. Biermer, in "Lehre vom Auswurf," gives the following analyses for the sputum of bronchial catarrh:

Water	97.994	97.798
Organic substances	1.369	1.743
Inorganic substances	0.635	0.457

Kossel ‡ discusses the method of determining the specific gravity of sputum, and finds that it increases with the pus-contents. In bronchitis it varies between 1014 and 1004.

Renk § gives two analyses:

Water, 98.3; solid constituents, 1.7; of which inorganic 0.53, and organic 1.17; of the latter, mucin 0.69, extractives 0.48.

Water, 97.04; solid constituents, 2.96; of which inorganic 0.76, and organic 2.2; of the latter, mucin 1.72, extractives 0.48.

The sputa contained almost no albumin and as little fat. On the other hand, Renk found fat occasionally in the sputum of phthisical patients, but not in that of bronchitics.

Petters has presented a communication on the sputum of bronchoblennorrhea.

Pain in the chest occurs in both acute and chronic bronchitis, but we can consider as really characteristic only the burning pain behind the sternum, which, with a sensation of tickling, introduces especially acute bronchitis, but is occasionally increased in chronic bronchitis. Pain in the intercostal muscles, in the epigastrium, and along the insertion of the diaphragm is certainly connected with, and increased by, the cough. But severe pain in these parts may also be produced by respiratory movements and by pressure. In a number of cases no pain at all is experienced even with the most violent cough.

Elevation of temperature is often present at the beginning of an acute bronchitis. The temperature rises at once. Chills, which may be repeated on the following day, are mentioned among the introductory symptoms; but the temperature quickly goes back to normal, even though the bronchitis is not at all improved. In the further course the fever may be again aroused, if an extensive capillary bronchitis leads to pneumonia. In chronic bronchitis there is no fever.

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* *Centralblatt für klin. Medicin*, 1894, Nr. 12.

† *Würzburger med. Zeitschr.*, vol. II, p. 336.

‡ *Zeitschr. für klin. Med.*, vol. XIII, 1888, p. 151.

§ *Zeitschrift für Biologie*, vol. XI, p. 103.

Headache is occasionally observed in the beginning of the disease, along with the febrile changes; and later, in chronic conditions, stases may occasion the same symptom.

Neuralgia of the head of peculiar severity during the initial period has been observed and described by Rilliet and Barthez in children, in whom they have also observed eclamptic attacks. In children and old people one must be prepared even for symptoms of stupor in the course of severe attacks of capillary bronchitis. The cyanosis existing at the same time points to beginning carbonic acid intoxication.

Insomnia, coated tongue, loss of appetite, vomiting (already mentioned), and **constipation** are often to be observed.

Urine.—No characteristic changes of the urine belong to the picture of an ordinary bronchitis; only when chronic forms lead to symptoms of stasis may these appear, and we will speak of them in their proper place.

The **circulatory apparatus** is in most cases, in strong and healthy people, intact, but there may be some increase of pulse frequency to correspond to the elevation of temperature. Irregular heart action occurs occasionally in the severe capillary bronchitis of children, but it is more frequent in the chronic bronchitis of old people, and then is certainly due more to the existing alteration of the heart than to the bronchitis.

VARIETIES.

As we do not possess enough observations regarding the characteristic course of individual cases to describe the forms given in the classification, and as this would lead to endless repetitions, it will be better to describe the different forms which are firmly established clinically. We may distinguish the following: *Acute bronchitis* of the larger air-passages (tracheobronchitis acuta); *capillary bronchitis* of children and old people (bronchitis capillaris); *chronic bronchitis:* (*a*) simple, (*b*) dry, (*c*) bronchoblennorrhea, (*d*) serous bronchorrhea.

Acute Bronchitis.—It begins usually with coryza and, in susceptible people, with febrile changes and a chill. The chill may be repeated and the temperature in children may even exceed 39° C. (102.2° F.). Pain in the head, hoarseness, a sensation of weakness, and loss of appetite may be added. The fever is not characteristic; there is no elevation of temperature in the morning.

The *cough* begins immediately; on the first day it is still dry, sounds exceedingly hollow and rough, and is not influenced by the position of the body. There may be a tendency to peculiar convulsive paroxysms of coughing, when the irritation is very great; these attacks attain great severity only in persons who do not take care of themselves at all and have no self-control, especially children, hysteric girls, and masturbating boys. The cough is accompanied by a rough and painful sensation behind the sternum; the point of bifurcation of the trachea is especially affected. On auscultation, we hear a few sonorous râles, especially between and below the shoulder-blades. If the cough is very violent, there is severe pain in the different portions of the thorax. This is caused by the straining of the expiratory muscles at their points of attachment to the thorax, not only the recti and obliqui, but also the pectorals, the scaleni, and the intercostal muscles. The most sensitive places correspond, usually, to the attachments of the first-named muscles That the trachea is especially sensitive to pressure, or even that cough may be easily excited by such pressure, I cannot assert. The discharge is purely mucous, scanty, and very tough; it is the *sputum crudum* of the older authors. In children these attacks of coughing may be accompanied by vomiting.

This frequently unpleasant and sometimes extremely distressing condition rarely lasts longer than two days; the condition becomes less disturbed, the dryness and roughness of the throat are alleviated, the discharge becomes more plentiful, large numbers of pus cells appear in it, and it takes on a more yellowish color (*sputum coctum*). In this sputum no desquamated alveolar cells are found, unless they were already present normally. This acute form of bronchitis is very quickly cured, but in a certain number of cases the catarrh drags on, and it may be so protracted that it quite loses its acute character and presents the picture of chronic bronchitis. Only in children and old people can the course be unfavorable, since in them a capillary bronchitis develops. [We agree with Forcheimer regarding the clinical course of streptococcal bronchitis: longer duration, greater fever, greater prostration, viscid expectoration and localization of râles and presumably of lesion. Any case in which streptococci are found with sputum will be prolonged, and in the old may prove fatal. Such prognosis, based on sputum examination, can be made before general symptoms would warrant. This has been our custom with great success.—Ed.]

Not rarely the cough shows certain regular peculiarities, which are difficult to understand; for instance, it begins at eight o'clock, when the patient first goes to bed, tortures him unceasingly, and disappears as by a stroke of magic at two o'clock, allowing him quiet sleep for the rest of the night.

Capillary bronchitis is not separated from the simple acute tracheobronchitis by any sharp boundary-line. It is not known how far the latter extends and what hinders it in many cases from becoming capillary; that is, from descending into the last divisions of the bronchi before the infundibula I believe that every extensive bronchitis becomes capillary; but in vigorous individuals, especially in adults, this causes no special symptoms, because the size of the respiratory surface is so great that a dangerous limitation of it can always be prevented by simple expiratory movements and by coughing.

In a certain number of individuals, however, this is no longer the case; decided disturbances of the gaseous exchange occur, and we speak of a capillary bronchitis, just as if it were a new disease. In truth, next to nothing has been proved concerning it.

We distinguish a bronchitis by continuity; that is, one in which the process begins in the larger bronchi and extends downward, and a primary capillary bronchitis, or one in which the pathologic process immediately attacks the small bronchi. As examples of the first kind we have the cases which begin with coryza and hoarseness; hence many cases of bronchitis from cold and the bronchitis of measles. For the second kind, we have as an example the bronchitis of typhoid fever.

Capillary Bronchitis of Children.—Up to about the third year, bronchitis assumes special importance on account of the fact that it causes considerable danger to life. As soon as the disease reaches a certain extent, a decided change of respiration may be noted; the rate is increased even to 60 respirations per minute; the single respiratory movements are peculiarly short and hurried, the alæ nasi are distended, and the lower part of the sternum is drawn in, as in stenosis of the larynx. Gradually the lips and face become cyanotic; continual attacks of coughing shake the little patient, but bring no relief. In great distress, he sits upright in bed, stares straight ahead with an anxious expression of countenance, he often draws his knees up to his chest, as if to support it, then lies down for a moment, then sits up again, then gets into his mother's arms, then back into bed. But gradually the apathy becomes greater, and a somnolent condition develops. For some time, when aroused from this condition by cold sponging, by cold affusions, or by the voice, he immediately falls back into the stupor, until finally the symptoms of edema of the lungs terminate the scene.

The breathing of these patients has been found very different; it is certain that there is a difficulty of inspiration and expiration, so that the inspiratory as well as the expiratory auxiliary muscles are called into action; in this the relations of the two to each other are not changed essentially. Biermer mentions that a type of breathing has also been observed in which expiratory dyspnea predominates, as in asthma. Concerning this I can only say that with great extension of the catarrh, the expiration has often seemed to me much prolonged. But such patients generally, as long as they are awake, breathe very irregularly; one must observe them in sleep, and in this condition I have not been able to demonstrate any marked slowing.

The dyspnea is not uniform or progressive, but is extremely variable; there are hours when it is quite endurable, and again hours when it reaches an alarming height. With the increasing restlessness the dyspnea also increases, and with the beginning of exhaustion it may diminish and sleep may ensue, refreshing the patient and introducing a turn for the better. Often, however, this improvement is only temporary and deceptive.

On inspection, attention is always attracted to the epigastrium. In quiet breathing, and, generally, when the air can enter the alveoli of the lung without difficulty, the epigastrium is arched during inspiration and depressed during expiration.

If the air cannot enter quickly enough and in sufficient quantity, the lungs are unable to expand and fill out the vacuum as fast as it is produced by the contraction of the diaphragm, and, as the latter cannot descend, the adjoining structures are drawn inward. In children these consist of the very elastic portions of the thorax, the lower end of the sternum, and contiguous portions of the ribs; hence retraction of these parts during inspiration is a sign that the quantity of air entering the bronchial tree is insufficient. It is a familiar symptom in diseases of the throat, and especially in stenosis of the larynx and trachea. The degree of retraction bears a certain relation to the size of the obstruction. If, in capillary bronchitis, the epigastrium is arched in the normal manner, respiration is sufficient; but the stronger the inspiratory retraction, the more wide-spread is the catarrh and the more extensive is the obstructed portion of the bronchial system.

Attention should also be called to the *distention of the lungs,* in the upper portions of the thorax. The supraclavicular and infraclavicular regions bulge and the respiratory movements become more and more indistinct until finally there seems to be no movement at all. The lung is forced to expand by the suction exerted during inspiration; and during the subsequent violent attempts at expiration, which accompany the paroxysms of coughing, the distention of the lungs becomes excessive. The air is drawn into the alveoli, but its escape is greatly impeded or prevented. It is easy to understand how during inspiration any obstruction in the bronchi may be overcome by the force of the inspiratory current, and that the air readily passes through collections of mucus, since the inspiratory movements do not tend to compress any portion of the bronchi, but, on the contrary, are accompanied by a distinct dilatation of these tubes. During expiration, on the other hand, the conditions are all much less favorable. It is true that if the alveoli empty themselves by their own elasticity, and the thoracic wall and diaphragm simply follow on passively, expiration is effected without any difficulty whatever. But if the thoracic wall and diaphragm attempt to empty the alveoli by active compression, it becomes at once apparent that they are not adapted for that purpose; for the pressure they exert on the alveoli must be very uneven—it affects not only the alveoli, but also the bronchi, which may be compressed here and there, and thus oppose the emptying of the alveoli. Such an expiration does not take place without stoppages and stases, which supply the conditions necessary for the development of distention of the lungs. There is no doubt that in severe cases, especially in children, this may lead to genuine emphysema.

The *percussion note* in children thus affected rarely presents very distinct changes; to prove dulness is very difficult and uncertain. Many authors emphasize a dulness in the lower portions, which appears at times and again disappears. This may be explained by the atelectasis, which generally occurs in the course of the disease and is not always a mere temporary condition, but may at times mark the transition from capillary bronchitis to pneumonia. It is an undoubted fact that small atelectatic areas are quite usually formed, but their demonstration by percussion and auscultation is much too difficult for practical purposes at the present time. Only when the areas involve the greater part of the lung is their demonstration possible through the slight dulness and weakened breathing over such areas.

Febrile changes are quite usually present in the capillary bronchitis of children, the temperature often reaching 39° C. (102.2° F.) and over. Unfortunately the fever of capillary bronchitis is quite irregular and is often entirely absent; it does not present a regular course, or, rather, in spite of the numerous observations, it is not yet clearly understood.

The *pulse* is much increased in rate, is rarely less than 120, and may reach 160; in that case it is very small, difficult to count and feel, and often dicrotic.

A more marked *cyanosis* of the face develops in the higher grades of the disease, and one can then gather from the face of the little patient the severity of the attack. The other portions of the body, especially the extremities, and the regions of the knees and elbows, are blue and cold.

A stage of great restlessness is followed by apathy and finally somnolence. Toward the end, *convulsions* are often observed. The most unfavorable cases usually terminate within the first fourteen days of the disease. But often there is a turn for the better, the breathing becomes freer, the pulse fuller, the signs of stasis disappear, the sputum is expelled more easily, and the râles diminish, the many small moist râles disappearing. But even in this stage an exacerbation of the catarrh or the formation of a pneumonic area may bring about an unfavorable termination.

[As the editor has previously expressed himself,* so-called capillary bronchitis is always infectious, and is in reality a broncho- or catarrhal pneumonia. This aspect will be discussed in the section on catarrhal pneumonia.—Ed.]

Capillary Bronchitis of the Old.—Bronchitis from cold, which occurs in old people, is one of their most frequent affections, and shows a very great tendency to extend to the tertiary bronchi. The disease gradually causes symptoms of exhaustion, which may rapidly increase and threaten life. Every attack of bronchitis in an old person is a direct menace to life. The character of the symptoms is not peculiar; especially can we say that in most cases the fever does not appear at all and that an afebrile condition is neither diagnostically nor prognostically of importance, and certainly not reassuring. The patients easily become somnolent. Although expectoration progresses quite favorably and the pulse is not weak, yet the patients continue in a half sleeping condition. From this condition they can be easily aroused, but if let alone, they fall back into it. The respiration is quickened, but not extremely so and not as in children. The inspiratory retraction of the epigastrium is not seen. At night there is often great restlessness; the patients sleep badly or not at all and in the morning feel extremely tired and wretched. At the same time they lose their appetite; the choicest foods are enjoyed in only the very smallest portions, and finally are utterly refused. On the other hand, the thirst is usually excessive, and the large amount of fluid taken then leads to a burdensome pollakiuria and polyuria, and the patients soil themselves, as small amounts of urine are often passed without their knowledge.

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* "Medical Diagnosis," 4th edition, 1900.

In those cases which last more than four weeks we cannot really speak of an acute bronchitis. Transition from the acute to the chronic form is quite frequent in old men. The more violent symptoms, especially of cough, gradually diminish, but the patient does not recover; expectoration continues to disturb his rest at night and the anorexia aids the increasing failure of strength. Marfan says that the chronic bronchitis of the old is almost always of cardiac or albuminuric origin. [In our experience it is a streptococcus infection most frequently.—Ed.] Such an assertion should, however, be accepted with great caution. It is, of course, easy to recognize an albuminuric form of bronchitis, and it is equally easy to assert, if this is not present, that the obstinacy of the bronchitis is due to the insufficiency of the heart. But it still remains only an assertion. The senile conditions which are always present to a greater or less degree in the hearts of the old are almost certain to carry with them the probability that the course of the bronchitis will be less favorable than at a more robust time of life. But so long as our considerations run along the line of such generalities, they remain arbitrary and practically worthless. The examination of the hearts of these old people gives quite a considerable percentage which show no abnormality at all; a strong, regular pulse of nearly normal rate (70 to 80) is presented in these cases. If toward the end, when the prognostic indications begin to be unfavorable, the pulse becomes irregular or increased or diminished in rate—the occurrence of a murmur is a rarity—it does not, in the author's opinion, justify the assumption of a cardiac form of bronchitis, but only of a bronchitis, which has taken an unfavorable turn on account of cardiac insufficiency.

As accessory symptoms which further increase the severity of the disease, we not infrequently see neuralgias, especially of the extremities and thorax.

The really dangerous complication, *bronchopneumonia*,—that is, the development of areas of infiltration in the lungs,—occurs so gradually that it entirely escapes diagnosis until on investigation these areas have become so large that they can be demonstrated both by auscultation and by percussion.

In many cases the question remains in doubt, and one man speaks of a pneumonia, where another would recognize only a bronchitis. The attempt to make the finest possible diagnosis may become dangerous in these cases, especially as the more positive and far-reaching decision is more generally approved.

The occurrence of lobar pneumonia is rare and is evidently to be expected only when there has been a specific infection. Such infections occur in adults and at the most vigorous time of life just as frequently as in the old. Cantani has very accurately described two family epidemics, in which the bronchitis led quite acutely to a pneumonia and, with especial frequency, to a pneumonia of the right upper lobes.* The cultures made by Manfredi gave no positive results. Sticker asserts that he has never observed contagious bronchopneumonia in Cologne.

Simple Chronic Bronchitis.—Many different conditions have been described under this name, but it is difficult to separate them, and in the confusion of observations one easily loses himself. It is usual to distinguish chronic bronchitis as an independent disease from bronchitis occurring secondarily in numerous conditions; but even in the latter case it may become so extensive and so important that the cause of it falls into the background, and even entirely disappears, while the result, the bronchitis, remains and becomes an independent affection. Accordingly we may distinguish the following forms of chronic bronchitis:

1. The forms which follow acute bronchitis; to this class belong also the forms occurring after measles, whooping-cough, and grippe. As the transition is evidently quite gradual, when shall we declare such a catarrh to be chronic? Gintrac has uttered the decision that a chronic bronchitis is one the duration of which exceeds forty days.

2. The forms which depend on constitutional anomalies (gout, rachitis, scrofula, and Bright's disease). To this class belong also certain forms observed in eczematous patients, which are so changed by the symptoms of the eczema that Gueneau de Mussy considered it necessary to give to this class a special name, "endermoses." We are glad to see the comparison of eczema and bronchitis used by the French, as it perhaps opens up an interesting field of study.

3. The forms which develop from chronic injurious influences. To this class belong by far the most important forms; many cases must be placed under this heading which men prefer to ascribe to taking cold, or to declare inexplicable. At the head of the list is the bronchitis of inebriates, then that of smokers, and to these may be added the form occurring in those who work in dust (called by Walshe "mechanical bronchitis").

4. The next group is made up of individuals who have some

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* *Centralblatt für klin. Medicin*, 1888, S. 508.

disease of the nose or nasopharynx which makes them breathe through their mouths and thus exposes the air-passages to injury.

5. Chronic bronchitis occurring in persons with circulatory disturbances: heart disease, obesity.

6. Chronic bronchitis in persons with any chronic disease of the lungs.

Generally the injuries under 2 and 3 have acted for a long time, causing several acute attacks of bronchitis, before chronic bronchitis develops. In children the injuries included under 1 are first to be thought of.*

Is there a syphilitic bronchitis? Sée cites Schlemmer as an author who says that by reason of the syphilitic endarteritis, which occurs so frequently, a bronchial catarrh may develop, which would therefore rank among the catarrhs due to circulatory disturbances, and presents nothing that is sufficiently characteristic to compel the diagnosis of specific bronchial catarrh. (Compare also the section on Syphilis, p. 173.)

Among the varieties of chronic bronchitis, that of childhood and that of old age have also been described as special forms; but the abovementioned enumeration of the etiologic factors includes those which are most important for these two periods of life, and a further subdivision of the subject would be of no advantage, but would rather tend to confusion by an unnecessary repetition of terms. The symptoms are, in general, those of bronchitis. The râles are so widely diffused and so variable in character as to be of little value. [When consonant, as the English writers term them, they suggest consolidations.—Ed.] Examination of the lung has merely a negative value by excluding consolidation and bronchiectasis. In other words, to guard against mistakes, the inferoposterior portions of the lungs, where small areas of dulness may easily be outlined by percussion, must be carefully examined, in order to decide the question whether a catarrhal pneumonia already exists or only a transient atelectatic or hypostatic condition. Such questions can often be solved by asking the patient to draw a deep breath and cough. Many cases, however, will remain doubtful; often one physician will diagnose catarrh where another deems himself justified in speaking of pneumonia. The latter diagnosis should be made only in the presence of well-marked and constant areas of dulness.

The *cough* is by far the most important symptom, but it is also extremely variable; sometimes it is infrequent and slight, at other times constant, violent, annoying, and even painful. Its severity is usually greatest in the morning and evening. A change of symptoms with the time of year and with the weather is quite usual, and perfect recovery is the more doubtful, the older the individual. The *sputum* varies as much as does the cough. The very tough and dry, the moist, the mucoid, and the purulent are distinguished, and between these are all possible transitional forms.

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*Comby, *Arch. gén. de méd.*, Nov., Dec., 1886.

Ordinary mucopurulent bronchitis is by far the most frequent form of chronic catarrh. The patients cough with very variable frequency, sleep poorly at night because the cough interrupts their rest, have a certain cachectic appearance, and in time become emphysematous.

Dry catarrh (*catarrhe sec* of Laennec) is very much more infrequent. Under what conditions it especially occurs is disputed; Laennec considers it the special catarrh of the gouty, but it certainly often occurs intercurrently during the course of the foregoing form. The mucosa is considerably swollen; indeed, the swelling at times is so excessive that Cantani has characterized the form as diffuse catarrhal bronchostenosis.* Then a marked dyspnea appears. There is also considerable cough, and as there is little discharge, the cough is annoying and wearing Here we often find severe pains at the points of attachment of the abdominal muscles to the lower portion of the thorax.

Fothergill, who was a sufferer from this affection, describes it as follows: An acute tearing pain is usually traced to one spot. This is a point of the periosteum, usually on a rib, where a muscle-fiber is torn off from its attachment by the violent strain of coughing. With every severe cough the growing reattachment of the fiber is lacerated, and this causes the stabbing pain. After some experience of this pain, the patient learns to assume an appropriate position when he feels the cough coming on. The position taken is one in which relatively little pull is exerted on the affected point during the act of coughing; in this way the laceration and accompanying pain are avoided. The hand is usually pressed firmly against the place, in order to lessen still further the strain on the young adhesions. Every one who has once experienced this complication of dry catarrh understands at once the significance of this position when he observes it in another. In the sputum we find numerous roundish masses of very tough mucus with pus cells and some blood-corpuscles (*sputa margaritacea, crachats perlés*). [The pearl-like masses of hyaline mucus usually contain Charcot-Leyden crystals and Curschmann's spirals.—Ed.]

Auscultation.—Moist râles are present in small numbers or not at all; but there is much piping (sibilant râles), accompanied by hissing, and sometimes by creaking, until finally a condition develops like that which is observed in asthmatic patients, where over the whole chest there is a piping, a creaking, or wheezing, which has given rise to the name "musical respiration." Often this catarrh is called by the laity and even by physicians asthma, especially when the dyspnea is very marked. Dry catarrh early leads to emphysema of the lungs and is frequently accompanied by it. Recently it has been asserted that it often occurs with dilatation of the stomach, but this association has not yet been satisfactorily proved.

The **serous form,** or *catarrhe pituiteux* of Laennec, is the rarest form of chronic bronchitis. In it we find a fluid, colorless, frothy sputum which has been compared to a thin solution of gum arabic. The cough is extremely violent and annoying, so that the patients become short of breath from coughing; hence the name *asthma humidum.* This form of catarrh is not dangerous, develops usually on a nervous foundation (nervous hypersecretion), but may become very annoying by reason of its unlimited duration. Usually, after a certain time, it passes over into the simple purulent form.

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* *Klin. Centralblatt,* 1885, S. 608.

Purulent bronchitis is the end-stage of every chronic bronchitis; besides there are certain forms in which there is from the first a marked production of pus. The sputum often seems similar to that of a phthisical patient; but when the secretion of pus becomes very abundant,—in twenty-four hours a discharge of pure pus up to 500 c.c. has been observed,—it is very characteristic, and similar to that of a bronchiectatic patient. This form has been especially distinguished as **bronchoblennorrhea.** The expectorated pus may at times take on a markedly offensive odor, but this is soon lost and is not to be compared to that of putrid bronchitis. Purulent bronchitis may, however, undoubtedly pass over into the true putrid form, especially when bronchiectases are present, but also when they are absent. These forms which are characterized by abundant production of pus also have a tendency to lead to irregular and at times even characteristic febrile changes; they then exhaust the patient and finally threaten his life.

The attempt to distinguish chronic tracheitis from chronic bronchitis must always appear forced. A pure chronic form of tracheitis must be something very rare. According to the description of systematic writers (especially Lubet-Barbon and Nicaise), the condition is characterized by the absence of all symptoms on the part of the lungs, and by a cough of peculiar timbre, which they call *timbre de chaudron fêlé;* tracheoscopy shows marked redness of the tracheal mucous membrane, and pressure on the uppermost ring of the trachea is very painful. Nicaise * has especially emphasized how the trachea is of great importance in the production of tone, and how, accordingly, tracheitis, as well as all diseases of the trachea, must have an effect on the voice.

The *course* of chronic bronchitis is neither dangerous nor painful in itself. There is nothing characteristic about it except the great tendency to recurrences, which is in a high degree peculiar to chronic bronchitis. Even a recovery, therefore, has rather the effect of a remission. The general health does not suffer, so long as no further complications are added.

In old people the course of chronic catarrh is frequently very grave. They are much troubled by the cough, and when expectoration is very abundant, the prognosis is especially unfavorable; although Lebert tells of a man who suffered for thirty years from such a catarrh and reached his eighty-second year. As a rule, the general condition suffers greatly. The appetite becomes poor, a tendency to edema develops, and in the evening there is an elevation of temperature. So the patients steadily grow worse, especially when the circumstances are not such as to permit the most careful nursing and all possible means of keeping up the strength. The disease usually passes over into the catarrhal pneumonia of the old; dull areas develop in the posterior and lower portions of the lungs and so they die. [The catarrh must be looked upon often as a terminal infection, whether in the course of some chronic inflammatory or degenerative process, or as the first break of life's chain in its senile decline.—Ed.]

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* Académie de médecine, 1891, 28 juillet.

Complications.—*Emphysema* should be named as the most frequent complication of chronic bronchial catarrh; every obstinate and frequently recurring catarrh eventually leads to emphysema, and the consequences of emphysema then gradually develop. In rare cases chronic forms of peribronchitis develop, and lead to cirrhotic changes in the lungs, or become the cause of bronchiectasis; they also prepare the most favorable soil for bacterial infection. It is difficult to say why in most cases emphysema develops, and in rarer cases chronic pneumonia. The latter is certainly more to be feared where the etiology shows violent injuries, which might lead to bronchopneumonia; especially the bronchitis from inhalation of dust is found in this class.

In a third group of cases the signs of a *circulatory disturbance* appear without the development of emphysema. The passage of the blood through the lungs is so retarded that stasis occurs in the right ventricle, in the right auricle, and finally in the venous circulation. We see, then, the whole picture of stasis as it occurs in emphysema and cardiac lesions; albuminuria, anasarca, ascites, hyperemia of the brain, with tendency to stupor and delirium appear, and the patient dies amid such symptoms as make the observer inclined to assume one of those heart lesions which do not reveal themselves by murmurs. In a number of cases emphysema lies intermediate between bronchitis and dilatation of the heart, but there are also cases in which this is not true, and in which we must assume a considerable injury to the pulmonary circulation directly due to the bronchitis. If one recalls the relation of the pulmonary artery to the small and medium-sized bronchi, then he will consider it as probable that the bronchitis can become injurious to the pulmonary artery only when it has led to a peribronchitis. Such forms as are found in connection with indurations and bronchiectases are most frequently accompanied by the most severe heart disturbances. They are, however, not the result of simple bronchitis, but must be attributed to the presence of some other specific infection. I believe that syphilis holds the first place

In old men who suffer from chronic catarrh and a certain grade of emphysema, nightly asthma-like attacks are observed, and Lebert has distinguished this asthma-like form of the catarrh of old age from the dyspneic paroxysms of the patients who suffer from emphysema and heart disease, as all the respiratory muscles are convulsively excited, the inspirations are especially difficult, the expirations easier and accompanied by sibilant and sonorous râles. A plentiful discharge ushers in an abatement of the symptoms.* I cannot regard these attacks as other than the paroxysms of coughing with dyspnea of bronchiectatic and emphysematous patients, which were mentioned earlier and have been called asthmatic attacks.

It is certain that there are forms of bronchitis with emphysema and dyspneic attacks which may just as well be described under the heading of asthma as under that of bronchitis or emphysema. I shall return to this subject under "Asthma."

DIAGNOSIS OF BRONCHITIS.

As in all diseases, there are also here, in addition to the cases which can be easily and certainly diagnosed, conditions which confuse even the most skilful observers. One should not forget that the diagnosis of the disease rests more on negative than on positive indications: Cough, some discharge, and some râles form the basis for our diagnosis, when nothing else is found which indicates another diagnosis. Hence the disease will frequently begin with the diagnosis "bronchitis," but in time everything else, even to the most severe diseases of the lungs, may develop from it. These possibilities will, as always, have to be borne in mind.

In the beginning of an acute bronchitis one will often have to consider the possibility of the case being one of whooping-cough or influenza. The former doubt is generally solved by further observation, but the second frequently persists, and the question might be asked with all seriousness whether many or all of our cases of bronchitis from cold are not really sporadic cases of influenza. Only by improving and facilitating our methods of etiologic investigation will it be possible to settle these questions, which are now quite unanswerable.

When the inflammation is limited to the trachea and largest bronchi, the râles may be entirely lacking. These cases are therefore designated tracheitis.

In many individuals, the cough has such a violent, *convulsive character* that it is hard to believe the case one of simple bronchitis. Although it is considered possible, it always seems to me doubtful; other possibilities, especially swelling and disease of the bronchial lymph-glands, at once suggest themselves, especially in children. A special section has been devoted to these conditions. Diseases of the larynx, of the nose, and of the pharynx, and foreign bodies which have found their way unobserved into the lungs, may cause a very similar convulsive cough; the diagnosis of bronchitis should not, therefore, be made in such cases until the upper air-

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*Lebert, *Klinik der Brustkrankheiten*, 1874, I, S. 102.

passages have been carefully inspected. It is also evident that the condition of the heart and large blood-vessels as well as that of the kidneys must be investigated and the sputum examined for tubercle bacilli before the diagnosis of simple bronchitis can be accepted.

It is especially difficult to draw a sharp line between *capillary bronchitis* and *bronchopneumonia* in children and old people; it has been said that this line is often drawn very arbitrarily. The diagnosis cannot be based on the fever alone; it requires the presence of an area of dulness. But as the areas of consolidation in these cases are often small and very circumscribed, dulness is very difficult to determine; and frequently one physician finds dulness where another refuses to concede its presence. The cases that lead to death, as a general rule, show considerable bronchopneumonic consolidation; but how about those that recover? Legendre and Bailly * make no distinction at all, but are of the opinion that the difference is merely one of form and degree. There is a catarrh of the capillary bronchi and alveoli, to which tissue changes are also added (so they say). According to whether one of the elements or the other predominates, the catarrhal pneumonia may assume one of two fundamental forms: the bronchial form, which is our capillary bronchitis, and the pneumonic form. Their efforts to keep these two forms separate only show that in the latter case there are well-characterized areas of dulness, while in the former this is not true, although on section small masses may be found scattered throughout the lung. We are making for ourselves difficulties which do not really exist in nature; the essence of the disease is always a diffuse capillary bronchitis. It dominates the picture, and the demonstration of areas of dulness does not materially affect our conception of the course, as is sometimes stated. It is believed that the prognosis may be more unfavorable when areas of dulness are shown than when they are not present, but it is a great mistake. The danger depends on the extent of the catarrh. The child endures a consolidated area, but when almost all the bronchi are so obstructed that an insufficient amount of air reaches the alveoli, the danger of suffocation is of course imminent.

The *latent pneumonia* of the old may easily be mistaken for a bronchitis; it is not practicable in private practice to subject such patients to a minute examination every day, and an area of dulness is easily overlooked; the exceeding weakness and the dry, red tongue are signs which must attract the attention of the physician.

As differential factors between *asthma* and the dyspneic conditions which occur in *chronic bronchitis* and, appearing spasmodically, may simulate asthma, I will mention the following: The asthmatic patient does not generally cough often and the attack may begin and end without expectoration. If the asthmatic patient has a laborious expectoration, he is surprisingly relieved by the expulsion of small amounts of sputum which has very characteristic peculiarities. The dyspnea of asthmatics is paroxysmal; that of bronchitics continuous, appearing even when there is no attack. The presence of acute distention of the lung would be just as decisive. But in the chronic forms of inveterate asthma the decision may be impossible. The layman will often speak of asthma, where the physician sees only emphysema and bronchitis.

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* *Archives générales,* 1844.

A warning is also necessary against confusing bronchitis with *hysteric cough.* The peculiarity of this is, however, not generally to be recognized; it is quite dry, or at least the discharge is extraordinarily scant; râles are not heard and the cough is of surprising violence, peculiarly harsh and barking, influenced by none of the ordinary remedies; it may last months, yes, even years, and be extremely annoying; but the cough disappears during sleep; it does not wake the patient; she must wake the cough. [The stigmata of hysteria are also present.—Ed.]

In the beginning of *tubercular affections* a dry cough may also occur, the recognition of which may present great difficulties. We get no sputum for examination, we hear nothing in the lungs; an evanescent râle at the apex seems no reason for awakening keen anxiety in the patient's mind and adopting far-reaching, costly therapeutic measures. How often may the physician impose disproportionate sacrifices, sow disquiet and excitement with their injurious consequences, where this is entirely superfluous. He can fulfil his duty only by attentive observation, and for this he requires the support of the relatives. A method of investigation which, according to my opinion, deserves to be considered, although it is now too much forgotten, is the determination of the respiratory volume by means of the Hutchinson spirometer. If the volume corresponds to the normal, one can refuse to be seriously anxious. [Tuberculin tests are justifiable.—Ed.]

When sputum is present, the diagnosis can, except in the rarest cases, be established by the aid of our present methods of investigation. We shall hold to the microscopic method of examination and shall not rely on the contradictory and uncertain diagnostic points given by the older authors.

The boundary-line between *capillary bronchitis* and bronchitis of the larger bronchi is often very arbitrarily drawn; we cannot speak of capillary bronchitis until we can satisfactorily determine the presence of small moist râles. If the number of râles which we can hear in a circumscribed spot, without changing the position of the stethoscope, is so great that we no longer hear two or three râles, but get the impression of one confused sound, in which we cannot count the different components, then we can, without further evidence, as Graves has already remarked, decide on the diagnosis of capillary bronchitis. [In capillary bronchitis or bronchopneumonia there is usually an excess of leucocytes.—Ed.]

If a chronic bronchitis lasts a very long time and no inflammatory symptoms are present, there is great probability that a certain degree of *emphysema* exists. If the diagnosis of emphysema is made at a time when distinct signs of enlargement of the lungs are obtained, mistakes will generally be avoided. In doing this, auscultation is of no assistance at all, for the classic diminution of the respiratory murmur only makes its appearance in the advanced stages of the disease, long after the differential diagnosis has been positively established by percussion; percussion and inspection are the methods to employ. We have to do with two conditions which belong together, and between which there is no sharp line. [The excursions of the diaphragm as indicated by inspection (Litten phenomena) and by the fluoroscope enable one to determine the presence of emphysema.—Ed.]

Under the name of the *night-cough of children*, Behrend described a condition in which the children do not cough at all during the day, go to sleep quietly at the usual time, but after one or two hours awaken, cough, cry, cough more and more violently, and finally vomit; the attack gradually subsides and finally disappears altogether; they fall asleep and sleep very well for the rest of the night. These attacks are repeated night after night for weeks, to the despair of the parents.* I have myself at times seen such cases, but am of the opinion that they are never due to disease of the bronchi or lungs, but to reflex irritation from the pharynx, from the nose, or from the cervical or bronchial glands. [Sir Andrew Clark pointed out the frequency of a similar cough in some forms of chlorosis.—Ed.]

TREATMENT OF CATARRH.

Prophylactic and Hygienic Treatment; Climatic Conditions; Mineral Springs; Potassium Salts and Sodium Carbonate; Tea, Expectorants, Balsams, Narcotics, Emetics, Strychnin, Astringents, Bleeding, Oxygen, Hydropathic Cures, Applications to the Skin, Baths, Inhalations, and Rarefied or Compressed Air.

Prophylaxis.—It is certain that by an intelligent manner of living the great majority of all catarrhs may be avoided; but if many lack the understanding for this, many also lack the energy and the means to change for the better their unfavorable conditions of life. Loud-mouthed reformers are wont to inveigh against faulty public hygiene, while they utterly neglect to enjoin the simplest precautions that every one could and should observe in his own home. The paramount importance of cleanliness, the need of which has become second nature to civilized man, and its power of protecting the community against a multiplicity of dangers, are far from being properly appreciated. That cleanliness includes a proper solicitude about the air we breathe should be obvious to everybody, yet the idea never occurs to most people. Education and training make up a second great factor. Rational hardening of the children is more and more practised even among the better classes, but the foolish fear of colds, with which hysteric mothers often maltreat their families all their lives, is still met with often enough, so that it is necessary constantly to reiterate its folly. The hardening process is entirely in the mother's hands, for she can begin with the proper training at the right time if she chooses.

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* Behrend, "Toux périodique nocturne des enfants," *Gaz. méd.*, 1846; Brunis, *ibid.*

The physician is called in after the catarrh has developed and, perhaps, become thoroughly rooted. He, too, must first of all turn his attention to hygienic measures.

[Certain simple measures sometimes succeed in aborting a threatening attack of bronchitis. These methods are based on diaphoresis and diuresis, and need no detailed description. Instead of the traditional quinin, liberal doses of the perchlorid of iron are strongly advocated by Owen.—Ed.]

We are accustomed to express ourselves briefly on the **hygiene** of bronchitis, because it includes a host of trivial matters. We are all agreed that the air must be pure, and yet in numerous cases there is no provision for changing the air in which the patient is confined. He must be protected from the evil influences of bad weather. To confine the patient to his room altogether is proper only in the first acute stage of the disease; this is the most radical method of protection, providing the air of the room is pure, evenly tempered, and contains the proper amount of moisture. [See papers by Forchheimer, Eicberg, and others on cold air in bronchopneumonia. —Ed.] But to continue this confinement for many weeks is contrary to our general principles. If the conditions are such that the patient must at all costs be protected from the cold,—as, for instance, in phthisis and in chronic catarrh with emphysema in elderly people,—he should be sent to a mild climate, if circumstances permit a journey. [But preferably not sent away if managed in accordance with the methods of Trudeau at Saranac in the Adirondacks.—Ed.]

In adapting the air to the needs of the body, more attention is now paid to the amount of **moisture.** It is certain that air that is too dry has an irritating effect on diseased bronchi, and may increase an acute inflammatory condition; air that is too moist has a depressing, relaxing influence, and diminishes nervous activity in inverse proportion to the temperature. In general, the relative amount of moisture which is to be regarded as most advantageous to the body is about 70%, and Clar * has contributed a short and readable paper on the method of regulating the amount of moisture in a room with the help of August's psychrometer. If at the normal room-temperature of 18.5° C. (73.6° F.) the difference between the two thermometers of the instrument reaches two degrees, the amount of moisture is approximately correct; if it is less, the amount of moisture is too great; if, on the other hand, it is more than two degrees, the amount of moisture is too small. The former condition is improved by ventilation; the latter by the evaporation of water in large vessels or by means of a spray.

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* *Blätter für klin. Hydrotherapie*, v, S. 63.

The longer the bronchitis lasts, the more necessary is it to attend to these important matters of hygiene and to summon to our aid some effective hardening methods. The simplest, the most effective, and the mildest form is a full cold bath in the morning. This can be begun with the smallest child, the temperature of the bath-water being moderated according to the time of year, beginning with 20° C. (77° F., better 90° F.) and using gradually cooler water. The influence of this cutaneous stimulation on the bronchi is evidently a very complicated process, in which reflex impressions on the vasomotor nerves no doubt play the chief rôle. If nothing more is gained, at least the skin will be brought into a condition to react vigorously to the unavoidable evil influences of the weather, and thus will protect all the internal organs from the circulatory disturbances which are the rule in delicate persons.

Care should also be given to the **clothing**; to adapt this to the prevailing weather is a precaution which children neglect from ignorance and adults from love of ease; in Germany woolen shirts and stockings are indispensable to catarrhal patients during the greater part of the year. The clothing should, in general, be warm and light.

If proper attention is paid to these general principles, it will be all that is necessary in many cases. The greatest difficulty in practice is to get a really **pure air,** as in our cities there is still much to be wished for in this direction. Many persons, by their occupation in dusty localities or dust-producing pursuits, are so constantly surrounded by dust that it is impossible for them to follow the physician's advice. But this class also can be charged with great neglect. How important it is to insist upon breathing through the nose, upon avoiding all unnecessary speech while doing such work and, when possible, warding off the dust by direct means of protection. There are various very effective respirators, which are recommended for this purpose; unfortunately many object to their use because it attracts attention, because it looks bad, or because to do something is not so easy as to do nothing. In a workroom the foolish make fun of the prudent; the sick frequently begrudge to the healthy their good luck, instead of taking a warning for their own benefit. We often hear in daily life remarks dictated by the—almost diabolical—idea, "Why need he be any better than I?" and there are invalids who think often enough "Why need he be healthier than I?" Such depravity is more common than many would believe, and should be taken into consideration by every prudent practitioner.

It has been justly said that the *hygiene of the cough* also demands careful attention in all chronic cases; and in some acute conditions, such as whooping-cough, this recommendation is highly important during the act of coughing. The chest should be well filled with air, the patient should sit or stand and steady himself with his hands. He should offer as much resistance as possible to the irritation which gives rise to the cough, and should try to shorten the attack. There is not the slightest doubt that cough is largely under the control of the will. Even in children an annoying cough has often enough been reduced to a minimum by suggestion. The patient should not sleep in a cold room or in a cold bed, or undress himself and change his clothing like a healthy person; his feet should be kept warm and any sudden change of temperature avoided.

The **diet** is a question the importance of which increases with the age of the patient. It is asserted that those who are inclined to emphysema bear fatty foods badly, while those who suffer from chronic inflammatory processes are said to derive great benefit from a diet rich in fats; hence cod-liver oil should be given to the latter, but not to the former. Alcohol is better for them; although the chronic bronchitis of emphysematous patients is often to be attributed to the abuse of alcoholic liquors. At this stage, however, its advantage is very great, and in proper dilution and amount, to be accurately prescribed by the physician, it is a safe remedy. In general, the diet should be regulated not so much by the bronchitis as by the general condition, the nutrition, the constitution, and the functional activity of the stomach.

In regard to **climatotherapy** * it may be said that for those who have the necessary means, the most appropriate climate should be sought out. In all cases of chronic bronchitis a mild sea climate is to be recommended for the winter months; the Isle of Wight, Lisbon and neighborhood, Madeira, and Teneriffe [Nassau, Jamaica, the islands of Bermuda, Florida, and southern California.—Ed.] are places to be considered. A certain relief for those who are not wealthy has been found in the use of a respirator, an instrument designed especially for those who have to work in dust, but which should always be worn out-of-doors in large cities by those who have a dangerous tendency to colds during the winter months. It is not to be recommended in all cases of bronchitis; it should not be worn by young people and is not adapted for those cases in which we still count on the advantage of active measures of treatment. But in the case of older people, where protective devices make up the greater part of the treatment, the use of a respirator has a distinct advantage.

Resorts nearer home are sometimes recommended, especially localities that are distinguished from the rest of Germany by their warmer climate and more protected situation. Wiesbaden, Baden-Baden, Würzburg, and Stuttgart have the reputation of being good resorts for bronchitics. There are also a number of smaller places in Germany that might be considered for these patients; they are generally well known to the physicians of the region. I will mention: Honnef in the Siebengebirge; Diez and Braunfels on the Lahn; Gleisweiler in the Palatinate; Weinheim an der Bergstrasse; the Muensterthal, and Gernsbach on the Murg, in Baden; Neustadt-Hohnstein in the Harz Mountains; Gruenberg in Silesia; Biesenthal and Eberswald near Berlin. These resorts are especially appropriate for a shorter stay during the summer vacation, a question that has to be decided much more frequently in ordinary practice. But any summer outing is good, if it only secures pure air. [Maine, New Hampshire, Canada, the northwest, in the summer; western North Carolina, South Carolina, and Georgia in the east; Arizona, New Mexico, and southern California in the winter, are the best regions in this country for bronchitic subjects to frequent.—Ed.]

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* See an instructive article by Bell, on "Experience in Search of a Cure for Asthma," etc., *Boston Med. and Surg. Jour.*, vol. CXLIV, No. 17, 1901.

The sea air deserves especial consideration; its purity and freshness, the strong movement of the air, and the high degree of moisture are of the greatest benefit to some constitutions. No influence is to be ascribed to the varying amount of salt. For young, vigorous individuals, in whom some weakness of the respiratory organs has remained after acute diseases of the lungs, the sojourn at the sea is, in my opinion, to be especially lauded. [During the winter months Atlantic City, Virginia Beach, and in dry forms of bronchitis, the east or west coast of Florida and the coast of southern California, are to be resorted to. The west coast is less enervating than the east coast of Florida.—Ed.] On the other hand, a high mountainous region is preferred by many. The mountain air is just as pure and fresh, the breezes are quite as stimulating, as at the seashore, but the air is relatively dry; and then the lower pressure of the atmosphere is to be considered. Thus we see that the demand on the lungs is greater in high mountainous regions than at the sea-level, but certain advantageous forces are present in the former that are lacking at the latter. In spite of these important differences, the choice of locality in the individual case is determined rather by the opinions and tastes of physicians and patients than by the comparative merits of the resorts under consideration. In general, it may be said that a high mountainous climate is better for people with phthisical habit and considerable anemia [as in parts of Colorado, Arizona, and New Mexico in the United States], while a maritime climate better suits emphysematous patients [except young persons]; in an overwhelming majority of cases the physician does not make his decision a question of conscience. The seashore and the mountains are both good for most patients.

The **mineral springs** are, in my estimation, to be regarded as climatic cures in the true sense of the word. Of all the methods of treatment of bronchitis, none is so extensively used as this, and it is unquestionably the most successful in all the more chronic cases. The number of places recommended for this purpose is very great: The most famous are: *Saline waters:* Homburg, Kissingen, and Soden. *Alkaline waters:* Gleichenberg; Liebwerda near Friedland in Bohemia; Salzbrunn in Silesia; Vichy; and Ems. *Alkaline-saline* waters: Marienbad; Rohitsch; and Tarasp. *Earthy:* Lippspringe; Weissenburg in Switzerland. *Sulphur waters:* Baden near Vienna; Schinznach; Nenndorf; Eilsen; Weilbach; and the Pyrenese spas. They belong to not less than five different groups of drinking-waters used in balneology, but I will not give the indications for them according to their chemical constituents, for not these chemical constituents, but the apparently secondary factors in the treatment, are really important. Life at a watering-place, which is usually beautifully situated and free from dust, with pure air and good food, far from the cares and anxieties of daily life, besides the use of active therapeutic measures, such as inhalations, which are carried out with greater regularity and for a longer time than would be possible at home—these factors are quite sufficient to explain the beneficial influence of these springs. If we wished to lose ourselves in the depths of speculation on the importance of sodium carbonate for the formation of blood, we might quote all kinds of experiments, the significance of which for the patient is practically *nil.* Such statements as the following, found in reputable works on balneology: Ems is especially adapted to the more torpid patients with relaxed mucous membranes and abundant secretion, Kochel for anemia, Weilbach for the scrofulous, Weissenburg for old chronic catarrhs with dry cough and scanty expectoration, etc., impress me as mere general expressions, in which we can scarcely find even a small kernel of truth. The situation, the presence of forests, the freedom of the air from dust, the average amount of moisture, in short, the therapeutic factors which I have already described as fundamentally hygienic, are also to be considered in the choice of a resort. The mineral springs themselves are practically of equal value. One can drink these alkaline or alkaline-muriated waters in larger amounts and with greater enjoyment; they suit human nature best, far better than pure water, and a small amount of sulphur in the water makes a pleasant and useful change. If the stronger purgative salts are present, their action, of course, becomes important; for corpulent patients and for those with a tendency to constipation and hemorrhoids, such springs will be especially preferred.

Of course, it is not a matter of indifference where one sends his patients, but the spring itself makes but little difference. The classification of mineral springs at present in vogue is too narrow and takes into account only their chemical constituents; we need books that shall tell us about the local physical and hygienic conditions in a thoroughly scientific way. The quickening influence of the recent advances in meteorology and hygiene is needed to stir the stagnant waters of balneotherapy.*

A special healing influence has been assigned to sulphur springs in cases of chronic bronchitis. Fromm says that he attaches value to sulphur waters only in those cases in which there is a tendency to hemorrhoids, with hyperemia of the liver. Weilbach, Eilsen, Nenndorf, and Landeck have been much lauded and are always eagerly sought by people with chronic bronchial catarrh. In still greater measure is this true in France with regard to the Pyrenese baths, Eaux Bonnes, Barèges, Cauterets, Bagnères-de-Luchon. In these latter places the climate also comes into consideration in great measure, as they lie relatively very high. The baths can exert no other influence than that of indifferent thermal springs. The amount of hydrogen sulphid contained in the spring is of moment only when the waters are taken at the spring itself. It is possible that the small amount of hydrogen sulphid which is excreted through the mucous membrane of the lungs after drinking from the Weilbach springs, for example, may be beneficial by causing a mild stimulation of the circulation in certain persons.

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* Balneological works, in which we would find out how the water is cared for at these watering-places, the sanitary conditions in regard to the disposal of sewage, how many typhus cases (enterica) have occurred in the last decade, and what ordinances, if any, exist for the protection of visitors, are badly needed.

In the most characteristic springs which patients with bronchial catarrh have sought for hundreds of years salt and sodium carbonate are prominent constituents. If we ask what these accomplish for the mucous membranes of the bronchial tract, when taken internally, we can answer with great positiveness: nothing at all. The good influence of plenty of salt on the general nutrition is established and will continue to assert itself at a resort as elsewhere. Of the influence of alkali, I can only say that it is generally recognized.

We can be certain that if they are taken with a sufficient amount of water, they will increase the secretion of mucus and render more fluid the mucus which is too tough. Unless an abundance of water is taken, their influence is as uncertain as that of the most worthless expectorant; the alkali alone has scarcely any value. I would not expose myself to criticism by drawing the naïve conclusion that the mucous membrane produces a more alkaline secretion which must favor the ciliary movement and facilitate the expulsion of the mucus. The essential point is the addition of fluid in a form which is agreeable and comfortable for the patient. These salts are also the best constituents to be added to waters for inhalation, and they owe part of their present reputation to that method of use.

One accomplishes the same by drinking *medicinal teas* as by drinking alkaline waters, and in chronic conditions this is by far the most useful means to be employed in ordinary practice.

In former times a number of different kinds of medicinal teas, which even now should be known to every physician as good household remedies, enjoyed considerable repute: Radix pimpinellæ, Fructus anisi vulgaris, Fructus anisi stellati, Fructus phellandrii; of these, the *species pectorales* (Althæa, Glycyrrhiza, Rhiz. irid. florent., Fol. farfaræ, Fol. verbasci, Fructus anisi stellati), the justly popular chest-teas, are still much used.

It is a matter of common experience that cough is relieved by the action of a mucilaginous medicament on the throat. These means can, of course, be really of use only in cases where the throat is in a condition of irritation. Under such circumstances, the succus liquirit., decoctum althææ [Flaxseed tea flavored with lemon; hot water with some spirits for the aged.—Ed.], and all the numerous cough syrups which are so highly prized, have a decidedly soothing influence. If one adds to them a trace of morphin, the sedative influence is naturally much greater, but the medication is also quite different.

We have now quite a considerable number of **internal remedies** which really act on the bronchi, either directly or indirectly, and we can say that the results must be due to an influence on the nerves, and, through them, on the secretion and circulation. Each drug has, however, its peculiar and complex influence, so that almost every one must be classed under more than one heading, for instance:

1. Medicines which act on the respiratory center: (*a*) Sedative: opiates and hydrocyanic acid. (*b*) Stimulating: belladonna, strychnin, ammonia, ipecacuanha, and senega.
2. Medicines which influence the activity of the glands: (*a*) Sedative: acids, belladonna, senega, and squills. (*b*) Stimulating: alkalies, pilocarpin, apomorphin, ipecacuanha, and tartarus stibiatus (red antimony) or tartar emetic.

The best we can do is to form natural groups, as far as possible, and discuss the various medicines in them.

A group which is always named in the first rank, but is very badly constructed, is the group of *expectorants*. An expectorant is a medicine which facilitates expectoration; hence the most various medicines may be included in this group; nothing is more arbitrary than the term expectorant. Niemeyer says that morphin is the best expectorant, and, under certain circumstances, he is right. In conditions of collapse, camphor and digitalis are expectorants. Reputable pharmacologists, however, include only the following drugs: ammonia preparations; antimony compounds; saponin preparations; apomorphin; ipecacuanha (Schmiedeberg); to which are often added the simple salts (common salt and potassium chlorate); the ingredients of chest tea (semini fœniculi, phellandrii anisi); and balsamic preparations (balsam of Peru, asafetida, galbanum—Buchheim-Harnack). In order not to increase endlessly the confusion, I will adhere to Schmiedeberg's classification.

1. *Ammonia preparations* have a stimulating effect on the spinal cord and on the medulla oblongata, and as small amounts of ammonia are excreted in the bronchi, they may favor the expulsion of fluid mucus by the irritation.* According to my experience, these medicines are of very little and very doubtful value, and are often injurious and objectionable.

2. *Saponin preparations*, at the head of which stands senega, cause irritation of the throat and an irritative cough—so much is certain; whether they increase or diminish the mucous secretion is doubtful, as we have authority for both statements † They are very bad-tasting medicines. Unless given in uselessly large doses

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* So Schmiedeberg thinks.

† According to Schmiedeberg, they increase the secretion; while according to Lauder Brunton, they decrease it.

they do no injury, and occasionally, when there are many mucous râles in the chest, a certain transitory improvement is gained.

3. *Antimony compounds* (stibium sulphuratum aurantiacum, SbS_5; kermes mineralis, or red antimony, a mixture that contains three parts of the sulphid of antimony; and tartar emetic, dose gr. $\frac{1}{16}$ to $\frac{1}{4}$) lower the blood pressure, as the blood-vessels are dilated; the heart action is not affected by therapeutic doses. The expectorant action is directly connected with the nausea and merely represents the preliminary stage of emesis; as this applies equally well to ipecacuanha and apomorphin, these together form a natural group among the expectorants. The stage of nausea is characterized by a peculiar increase in the secretion of mucus and saliva. A moderate increase may thus be maintained for a long time, and so this medicine is useful in a less degree than, but in the same way, as pilocarpin, which is nearly related to this group of expectorants, and which I regard as the best.

In ordinary practice ipecacuanha is preferred; its action is slow and quite uncertain, and it is entirely without danger.

Apomorphin,* which is more certain in its action, may be given as an expectorant in doses of 0.05 (gr. $\frac{3}{4}$); this may be rapidly increased to 0.1 (gr. $1\frac{1}{2}$), and even 0.15 (gr. $2\frac{1}{4}$), three times a day. Apomorphin also has the advantage of being absorbed when given in inunction. It may be advantageously combined with morphin.

[The doses in the text are excessive; for adults $\frac{1}{10}$ of a grain is the usual dose, which may with safety be increased to $\frac{1}{5}$. Ewart gives gr. $\frac{1}{30}$ hypodermically for children.—Ed.]

The most dangerous and most certain of these medicines is *pilocarpin;* it is excellent when used in minimal doses, and then I have never experienced any injurious effects from its use (0.0005 to 0.001—about gr. $\frac{1}{125}$ to gr. $\frac{1}{60}$—three to five times a day). All these medicines are to be used only for a short period at a time, for after a few days the organism becomes accustomed to them and they cease to act. Besides, they are to be used only when the sputum is very tenacious and scanty; hence in dry catarrh and in the first stages of acute catarrh. They can also be used for a short time in chronic catarrh, when the patients complain of dry, painful cough. I consider them superfluous in most cases; we can accomplish quite as much by ordering teas and alkaline waters. The convenience of administration, however, will always assure them a large following.

Among sedatives, belladonna, hydrocyanic acid, and morphin are usually mentioned, but the last is really the only one which is certain in its action. [Heroin and other opium preparations also have their field of usefulness.—Ed.] Belladonna has been found surprisingly useful when inhaled with the spray. Cocain may also be mentioned in this connection; its effect is, however, due to a local influence on the irritable zones of the larynx and trachea, hence it brings about a purely symptomatic alleviation of the violent, irritating cough. If we should give belladonna until there began to be a dryness of the throat, the patients would complain most bitterly. After the use of morphin, the secretion becomes scanty, and the cough more troublesome; this is the case when we give too large doses, but if minute doses (0.001—about gr. $\frac{1}{60}$—at most) are given, such complaints are not heard, and the quieting effect (infrequent, alleviated cough with abundant expectoration) is surprising.

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* The drug turns green when exposed to the air, but does not lose its efficacy.

Potassium iodid is a reliable stimulating remedy for the mucous membranes of the respiratory tract. It is excreted in small amounts from the mucous membranes and has a stimulating effect on their secretion. Hence it is unquestionably an expectorant, although it is not usually described among them. Its usefulness is very uncertain and varies in different individuals. The good effects obtained in asthma do not justify us in extending its use to cases of bronchitis. It may always be tried, but the definite and rational indications for its use cannot yet be given. It is effective only by way of favoring the secretion; pilocarpin would certainly be preferable.

In the books the *balsams* play a not inconsiderable part, and at the head of these stand the balsam of copaiba and turpentine. [Terebene and oil of eucalyptus may be added.—Ed.] It is always asserted that they have the effect of diminishing the secretion. As a matter of fact, this influence is one of the most unreliable, and cannot be utilized. It is certain that volatile constituents of these bodies are excreted with the current of air. Nothing positive is known in regard to their influence on catarrh of the respiratory tract; and as these drugs have unpleasant effects, I have discarded them altogether. Still less can we boast of the results obtained from the use of tar, which is sometimes recommended, of balsam of Peru, asafetida, galbanum, and oil of eucalyptol. Limitation of the secretion is certainly brought about by the use of atropin and kindred drugs, and therefore these may be used for a time symptomatically, but no healing effect is to be expected from their use. The means which are really of use in these cases are cold sponging, salt-water baths, and inhalations.

It is easily seen that I regard the usual medicinal treatment of bronchitis and the conditions related to it as pure empiricism; but that I may not be charged with expressing only one-sided views, I will give the opinions of three noted authors, one German, one English, and one French:

Eichhorst employs for increasing the secretion, ammonium muriate with succus liquidus; apomorphin; ipecacuanha; and, most of all, potassium iodid. Among remedies that assist the mechanical expulsion of the secretion, he praises ipecacuanha, stibium sulphuratum aurantiacum (red antimony), and decoctum senegæ with liquor ammonii anisatus.

Fothergill distinguishes:

Relaxing expectorants: tartarus stibiatus (red antimony), aconite, potassium iodid, ammonium acetate, ipecacuanha; and assumes that they dilate the blood-vessels of the body and by this means relieve the distention and swelling of the bronchi and stimulate their secretion.

Stimulating expectorants: squills, strychnin, ammoniac, belladonna, senega, and benzoin. "Never give squills until the skin is moist and the mucus is loose," says an old book on practice. They act as stimulants on the respiratory center; so he says of strychnin that it is useful in acute bronchitis, when the expectoration is difficult; in chronic bronchitis it facilitates the laboring respiration, and when the right ventricle is dilated, it increases the effectiveness of digitalis.

Ferrand says: "The remedies which are designed to favor the expulsion of the secreted product are called expectorants."* Most of these are capable of influencing these products, of making them more fluid, and of exciting contractions either of the bronchial muscles or of the strong auxiliary muscles of expiration. Among the former he includes acidulous beverages fruit bonbons, nitrate of sodium and potassium, polygala, squills, gummi ammoniacum, jaborandi; among the latter, the emetics and nauseating drugs. He also claims to influence the mucous membrane so that it stops pouring out a superabundance of secretion into the bronchi, and he expects this result from the use of the balsams, the sulphur preparations, and, finally, from the preparations of antimony (kermes mineralis, sulphurated antimony, and tartarus stibiatus). Of potassium iodid he says that it passes through the superficial portions of the mucous membrane and produces a slight modification of the catarrhal secretion, which changes the mucosa of the bronchi and makes it less sensitive to external influences; it also makes it less fitted to remain relatively dry. The antimony preparations act in a similar way. After learning that they limit the secretion, we are now told that they increase it!

There are, unfortunately, cases enough in which the cough is so annoying that it becomes necessary to use remedies that *lower the irritability of the nerve-centers;* this is best done by opium and morphin or heroin, but chloral and almond water may also be tried. In many cases, in spite of the external quieting, these remedies cause a marked degree of cyanosis; I would advise, according to the proposition of Davies, that before their use 0.05 (gr. $\frac{3}{4}$) extract of belladonna be dissolved in 15 c.c. of distilled water and inhaled.

If the mucus accumulates to such a degree that it must be removed at any price, a few doses of one of the above-mentioned *emetics* may be given. If the mucus accumulates again, after it has been removed by an emetic, it is a serious warning not to continue the treatment, but rather to stimulate and strengthen the cardiac and respiratory nerves by cold sponging and cold shower-baths; in very bad cases, wine and camphor may be taken internally, but no emetics should be given.

As a therapeutic curiosity, the knowledge of which I owe to Fothergill, I will mention the use of strychnin. He calls attention to the fact that cases of dyspnea which occur so frequently at night in old bronchial catarrhs, are the consequence either of insufficient heart action or of insufficient irritability of the respiratory center. In deep sleep the breathing in these cases becomes so weak that the blood becomes loaded with carbonic acid, and when this reaches a certain point, the attack of dyspnea comes on. As soon as the blood is purged of the carbonic acid, the patient sinks into a quiet sleep; but if the right ventricle suffers, the attack is not so quickly relieved, and may last for some time. For the first case, strychnin as a "respiratory stimulant" is the right remedy to use I quote this passage *in extenso* because I do not wish to criticize it, but consider that the collection of further therapeutic observations is necessary. Duncan has described a case of very marked expiratory dyspnea, which was wonderfully improved by the use of pills containing nux vomica 0.03 (gr. ½) and ipecacuanha 0.06 (gr. j).*

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* No author has hitherto been able to give us a better definition.

If I have occasionally indulged in disparaging remarks about these remedies hitherto mentioned, I may say in regard to the following group that I consider all its members wholly objectionable. These are the *astringents*, at the head of which stand tannin and lead.

It is interesting that **bleeding** is now and then recommended in the treatment of bronchitis. Fothergill mentions a case as a curiosity. Ferrand, however, gives it a decided encomium: "Venesection (in inflammatory bronchitis) is a powerful remedy and, if not absolutely counterindicated by the general condition, may have a wonderful influence. It reduces the beginning stasis, lowers the blood pressure (?), and, by making room in the chest for the entrance of air (?), favors the process of hematosis." I believe myself that such a powerful remedy will, under certain circumstances, exert a very decided influence; but under what circumstances will that influence be curative?

[In acute suffocative bronchitis, "if the case do not improve it is rapidly deteriorating, and at any moment, owing to progressive congestion of the heart, exhaustion may set in. Our duty is to apply the only adequate remedy, *venesection*, without waiting for the manifestations of extreme cyanosis, cold sweats, jactitation, and fluttering pulse." The removal of 8 to 10 ounces from the external jugular vein is advised (Ewart).—Ed.]

Just as little as for bleeding has any useful indication for the *inhalation of oxygen* been presented, although some wonderful results have been reported from its use.

After all the remedies mentioned, I will now speak of the **hydriatric method,** which is the best and most effective of all. According to my experience, every form of bronchitis is favorably influenced; and if it does not cure, then either other methods do not, or else we have before us a special form, which needs special therapeutic measures. The simplest form in which cold water is here to be used is a *Priessnitz compress*, which may be made large enough to envelop the whole thorax. The frequency with which this is to be changed must be regulated by the physician, after due consideration of the patient's strength and the urgency of the indications.

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* *Dublin Quarterly Journal*, 1860.

Next in simplicity are *cold affusions*, the patient standing or sitting in lukewarm water; they play the first rôle in pediatric practice on account of the ease and convenience of administration. This is therefore the best method to use in the capillary bronchitis of children.* It replaces in this disease almost all other applications that are found in the writings of the older physicians in even greater profusion than in the works cited in the bibliography of this article, which give only a faint idea of all that such children had to submit to in former times.

As the third method, may be named the *cold wet pack*, which is the best for nervous and excitable patients. In the case of vigorous individuals *cold rubs* or the *cold douche* is the most energetic and the best method of applying water.

In special cases we can make use of *warm baths* or even of *vapor baths*. In the very early stages of acute bronchitis, vapor baths, with subsequent sweating, may be a very advantageous introductory treatment; in the chronic forms, on the other hand, warm baths of long duration are decidedly better. I must confess that I consider hydrotherapy the method which, if rightly applied, will accomplish the greatest amount of good in all cases, and only in very extreme cases will it be necessary to call to our assistance for a time the stimulating or sedative medicines.

Local Applications.—Besides the simple application of water, the external skin has been treated in other ways for this affection: The most varied poultices, paintings, and inunctions have been used. If we read the book of Ferrand, we see what an important rôle the French assign to these measures; dry and wet cups, vesicants, alcoholic and ammoniacal inunctions, painting with tincture of iodin, large mustard plasters, are recommended one after another and highly lauded. The milder of these means may often be necessary, especially when the authority of the physician needs for its support the continual occupation of the patient. Poultices must be employed with great caution; I have seen their use followed by unpleasant burns in old persons; and, besides, they are heavy and troublesome. I believe that a good Priessnitz compress with wax paper [or rubber cloth] best satisfies all reasonable demands. To carry the irritation of the skin further than the formation of a small, recognizable miliary eruption, which can be produced by the diligent use of water applications, I consider superfluous, and objectionable on scientific grounds. Nevertheless there are physicians and patients who are enraptured with tartar emetic ointment and with croton oil liniment. When a fine pustular eruption covers the chest, they have at least something to look at, and the patient will not worry about the lack of treatment. That there are people who must be treated in this way is undeniable; and we are often forced to employ measures which are practically excellent but scientifically worthless.

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* From a distinguished French work of very recent date I extract the following quotation on the treatment of the bronchitis of children, in order to warn most seriously against such therapy: In the severe form one should at first give quinin sulphate or antipyrin. In very young children these remedies may be given in the form of an enema or in an inunction (in the axilla!); then revulsives will be used. Mustard plasters are used alternately with turpentine liniment. When it is necessary, we should use expectorants, ipecacuanha, kermes, ammonium acetate or benzoate, apomorphin. (A long encomium and direction for its use follow.) When the children are older than five years, one can give the sedative remedies in small doses. Caffein in subcutaneous injections, 0.05 (gr. ¾) daily, divided into several doses, is of good service in children of one year, when the catarrh threatens to change into a capillary bronchitis.

The treatment of true capillary bronchitis I shall also reproduce for the benefit of the friends of polypharmacy; I consider it, of course, as bad and as improper as the preceding: The feet are packed in cotton and gum-taffeta and sinapisms or dry cups are applied to the chest. Every hour a spoonful of the following mixture is given: Ammonium acetate, 0.5 to 1.0 gm. (gr. viiss to xv); syrup. codeini, 10 to 30 c.c. (f ʒiij to f ℥j); tincture of aconite, gtt. xv; potio gummosa, 100 c.c. (f ℥iij). If the case is severe, a lukewarm mustard bath should be given for five minutes. One can give an emetic, but it should not be repeated. If the disease is fully developed, then we should lay an epispastic for three hours on that part of the chest where auscultation shows the greatest changes; this is covered with a dressing of cotton and borated vaselin, on the top of which a paste of potato meal is applied. One can after two days repeat the epispastic. Increasing weakness calls for coffee or champagne with water or alcohol (this is really a good measure). If the patient is delirious, then we give him an enema of chloral, 1.0 (gr. xv); tr. moschi, gtt. xx; tr. valerian, gtt. xx; the yolk of one egg; and water, 150 c.c. (f ℥v). To regulate the temperature and circulation we can give each morning 0.15 (gr. 2¼) of quinin sulphate. Against oliguria (consequence of the asphyxia and cause of the increase of the bronchial affection) we should use powder of digitalis.

A quite decided and curative indication will often be fulfilled in acute cases if we can succeed in getting the patient into a *perspiration.* Copious drafts of hot tea prepared by the skilful housewife from the juice of the black currant [box-berry], and plenty of blankets, will easily accomplish this object without resorting to the use of medicines. The bath proposed by James Simpson does not seem to me bad under simple conditions: A number of soda-water flasks are filled with hot water and well corked. Stockings are wrung out of hot water and drawn over the flasks, then these are placed around the patient and between his legs, and he is well covered. He soon finds himself in a kind of vapor bath and begins to sweat.

Inhalations.—Judging from my own experience, inhalations are next in value to the hydrotherapeutic measures. Even the simple inhalation of warm water vapor from a tea-kettle is extremely grateful to the patient, but the proper way is to use the old (Siegle's) inhaler, with physiologic saline solution for the fluid. The older and more chronic the cases, the less will be accomplished by inhalation, but it is always a useful measure. When there is dry cough and marked swelling of the mucosa in acute cases, it not infrequently checks the progress of the disease. To increase the efficacy of this method innumerable inhalation fluids have been recommended; beginning with Ems water, we find all imaginable recommendations. Alkalies in the most varied concentration, with the addition of extract of belladonna and cocain in proper dilution, to allay irritation, seem to me at times quite efficacious. [Inhalations of the vapor arising from hot water

(Oj) to which turpentine (two drops), tincture of benzoin (ʒj), or creosote (gtt. j–ij) are added.—Ed.] As a rule, I gain the desired results with the saline solution alone; it quiets the cough, relieves the pain, and checks the mucous discharge. The inhalations would win greater respect if they were used in the earlier stages and not in the old and protracted cases which demand from them what no medicine is able to accomplish. In all severe and older cases the inhalations should be used more frequently and for a longer time; every two hours, and ten minutes at a time, is not too much.* The inhalation method has been considerably advanced by the introduction of *cabinets*,† in which the whole atmosphere of the room is filled with a very finely divided aqueous vapor; the patient practically breathes in a mist. These minute particles of water unquestionably reach the bronchioles and alveoli. However, observations on the results of their use are lacking. [See Ringer and Murrell. Inhalations from steam atomizers of tar, and wine of ipecacuanha are of great service.—Ed.]

Inhalations gained a certain temporary popularity thanks to *Waldenburg's apparatus.* He recommends consecutive inhalations of very slightly *concentrated air* (increase of $\frac{1}{20}$ atmosphere). We still see the method used in some places,—for instance, at Ems,—but it has almost disappeared from ordinary practice. We have no longer the slightest doubt that in this method the higher pressure of the air has nothing at all to do with the results, for improvements occur often enough to make the method appear worthy of respect; it is really only a form of respiratory gymnastics, and the method should be replaced by simpler and more effective gymnastic exercises. For a time it became so fashionable that the most detailed investigations are found in the literature. If any one is interested in these almost worthless matters,‡ he will find them in Riegel's writings.

Respiratory gymnastics are best carried on where the air is pure and entirely free from dust, as in the forest or at the seashore. Certain motions of the arms with and without dumb-bells or a staff, deep inspirations, and very long and systematic expirations will prove effective and healthful measures to any one who carries them out with patience and persistence.

After a thorough analysis, my treatment of broncho-catarrh will be found extremely simple.

In *acute bronchitis:* In the ordinary cases, inhalation of nebulized physiologic saline solution, and at the beginning some pilocarpin; later a mild narcotic if the cough is painful, and Priessnitz's compress around the throat and chest, changed at short intervals or used only at night. In severe cases, especially in the capillary forms and in children, active hydrotherapeutic measures should be begun immediately. In conditions of weakness wine, ether, camphor, and beef-broth may be used. In the old, the use of hydrotherapy is always attended by great difficulties; in these cases we should like to have remedies which we could trust. Nevertheless I must give a special warning against the use of expectorants. Priessnitz's compresses, changed every two hours, wine, and, if the night's rest is interrupted, opiates are the best treatment for them. If the conditions permit it, a warm bath twice a week, with cooling douche, has a very refreshing effect.

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* Recently a so-called Norwegian apparatus has been presented with no small claims. It is not to be recommended.

† In Ems and Dresden.

‡ Criticism by Josephs: "Wirkungslosigkeit und Nachtheile der transportablen pneumatischen Apparate von und nach Waldenburg," Hamburg, 1877.

In *chronic bronchial catarrh* the therapeutic indications are much less definite than in the acute, and in these cases it is often necessary to try different methods. A hydrotherapeutic measure adapted to the individual case is, in my opinion, the best of all methods; this may be combined with the use of alkaline or sulphur waters in large amounts. The attempt to bring about further improvement by sending the patient to a watering-place is always to be urgently recommended in summer and under favorable circumstances. The mere sojourn in the country is decidedly helpful in many cases.*

If a constitutional anomaly exists, it must of course be attended to first; but it is not advisable to treat a case of bronchitis in a syphilitic subject with iodin or mercury, unless ulcerations are present in the bronchi, or infiltrations in the lungs. The use of iodin is rather apt to produce the catarrh and to make it more persistent. For these forms, the best means of relief are pure, fresh air, a mild climate, and the use of the inhalation cabinet recently adopted at Ems and Dresden.

When the air-passages are affected by some external injury, the first obvious indication is the removal of the cause, as has been mentioned. Conditions interfering with nasal respiration require especial attention, and the patient should consult a specialist in regard to the advisability of operative interference.

Those in whom there is an injury to the circulation may expect the greatest benefit from the use of tonics and cardiac stimulants. They should take digitalis from time to time, exercise freely in the open air (mountain climbing), and exert themselves otherwise in gymnastic exercises, especially in lung gymnastics and in respiratory exercises; they should also sponge the chest and the whole body with cold water.

The corpulent are also subject to catarrh on account of insufficient circulation. Here the treatment of the obesity is to be taken directly into account, in addition to respiratory and cardiac gymnastics.†

The *catarrh of inebriates* occupies an especial place, in so far as it is always dependent on circulatory disturbances; the measures just recommended are therefore to be especially considered in this condition. In doing so the constitution is to be regarded and the abuse of alcoholic beverages replaced by a reasonable indulgence. These indications can be satisfied best by a sojourn at the seashore. But aside from the cases of inebriates, the indications are not always simple, and it often becomes necessary to supplement or assist the treatment by a stay at one of the different health resorts. The above material, learned from experience, will serve for general principles.

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* Consult Greenow on chronic bronchitis.—Ed.

† See Anders: "Obesity and Bronchitis," *Penna. Med. Jour.*, Feb., 1902.

The forms that coexist with other diseases of the lungs may present peculiar difficulties on account of the reciprocal effect of the two diseases on each other; the bronchitis, on the one hand, increasing the severity of the lung disease, and, on the other, being itself aggravated by the pulmonary affection. In these cases it is the lung diseases that should be treated. It is true that in emphysema we are inclined to treat the bronchial catarrh because it seems easier to attack than the emphysema, which quite commonly depends on the catarrh and is directly caused by it Nevertheless the treatment of the accompanying bronchitis is of far less value in emphysema than the treatment of the emphysema itself, which will be considered in another place.

[The simplicity of the author's treatment of bronchitis cannot fail to appeal to every one. Certainly no safer, more rational, or agreeable method, and one more effectual, can be carried out, as any one with practical experience will testify. Unfortunately there are cases and cases. To the lay mind the larger proportion of cases do not require so-called coddling treatment, hydrotherapy, etc. The American laborer and business man will not stop work for that which they deem a minor ailment, although they may experience the contrary.

We see cases

I. Beginning with an acute "cold," coryza and perhaps pharyngitis, with general symptoms of malaise, fever, and headache and muscular pains. Experience has taught these patients that a bronchitis is likely to follow if the "cold" is not aborted.

II. Cases with laryngitis which rapidly extends to the trachea and bronchi.

III. Primary acute bronchitis.

The first class of cases is to be treated like acute coryza with general measures, as recommended by the distinguished author; with a brisk hydragogue cathartic, as elaterium, if the patient is robust; or with a full dose of Dover's powder at bed-time, and a hot foot-bath. It is in these cases that Bulkley recommends 20 grains of bicarbonate of soda in half a glass of water every fifteen minutes for two or three hours. Others recommend small doses of aconite, a remedy I never employ. More effective are camphor and belladonna. Some one of the following prescriptions will meet the indications presented by individuals. If there is much headache and frontal pain, the first may be employed.

℞.	Camphor	gr. j	0.06	
	Extract of belladonna	gr. j–iss	0.06–0.09	
	Quinin sulphate	gr. j–ij	0.06–0.12	
	Morphin sulphate	gr. $\frac{1}{40}$	0.0015.	M.

Ft. Caps. No. j. Directions: One every half hour for four doses; then one every three hours.

If the skin is hot and dry and there is restlessness, one grain of Dover's powder may be substituted for the morphin. If the patient is robust and the headache intense, acetanilid, grain j to ij (0.06 to 0.12), may be given with the ingredients of the first prescription. It is obvious that these remedies are to be given in the late afternoon or evening when the patient is confined to the house.

In addition, the usual antiseptic washes which relieve the congested nostrils may be employed, as boric or carbolic acid lotions, alkaline lotions, cocain carefully, or suprarenal extract.

In the second instance we must abort or assuage symptoms by the general methods which the author recommends, to which I add the cold throat compress at night, the use of inhalations of the vapor of compound tincture of benzoin in hot water, or of carbolic acid in hot water, using an inhaling bottle for the latter. Here, again, a purgative and a diaphoretic are timely.

In the third class of cases we are guided by the arbitrary division of the dry and moist stage of the bronchial catarrh as indicated by the expectoration. In the first stage, in addition to measures recommended by the author, or, in their stead, if they cannot be employed, we are accustomed to use the following combination:

If the patient is going about, suffering with myalgia, chest oppression, tight cough, often harassing, we may select, first—

℞.	Quinin sulphate	gr. j–ij	0.06–0.12
	Dover's powder	gr. j	0.06
	Acetanilid.	gr. j	0.06
	Or—		
	Salicin	gr. j	0.06.

Caps. No. j. Directions: Two at once, two in two hours, and one every three hours.

Or—

℞. Mist. potas. citrat.
One tablespoonful every two hours.

Or—

℞.	Potassium iodid.	gr. xlv	3.0	
	Wine of ipecac.	ʒ ij	10.0	
	Brown mixture	q. s. ℥ iij	100.0.	M.

Directions: One teaspoonful every two or three hours.

If the cough is harassing, $\frac{1}{30}$ to $\frac{1}{12}$ (0.002 to 0.005) grain of heroin may be added to the dose in any combination.

Though nauseating, it is sometimes necessary to use the following before relief is given to the cough and oppression:

℞.	Syrup of ipecac			
	Syrup of sanguinaria			
	Syrup of squills	āā ʒ ij	10.0	
	Syrup of wild cherry	q. s. ℥ ij	60.0.	M.

Directions: One-half to one teaspoonful every two or three hours.

In the treatment of the next stage we differ in some respects from the author in that we find chlorid of ammonia, terpin hydrate, terebene, and tar preparations, singly or combined, of much value. Thus:

℞.	Ammonia chlorid	ʒ j	4.00
	Terpin hydrate	ʒ j	4.00
	Dover's powder	gr. xxiv	1.50
	Or—		
	Heroin	gr. ss	0.03.

M. ft. caps. No. xxiv.
Sig.—One or two (according to age) every two or three hours.

If the cough is easy, the expectoration free, the opiate can be withheld. If there is exhaustion, digitalis or strychnin may be employed in suitable doses. Terebene in capsules or on sugar may be used. Tar (pix) in doses of two grains may replace the opiate or the terpin hydrate. Many of my patients keep in their medicine chests the following:

℞.	Ammonium chlorid	ʒ j	4.0	
	Wine of tar	℥ iss	50.0	
	Brown mixture	℥ iss	50.0.	M.

Sig.—Two teaspoonfuls every two or three hours.

Sometimes iodid of potassium is added.

It is remarkable that stimulants, as strychnin, keep up blood pressure and arterial congestion and thereby aggravate any bronchitis present. Again, we are justified in the use of such remedies if venous instead of arterial hyperemia appears to exist.—Ed.]

FIBRINOUS BRONCHITIS.

For the literature up to 1876 compare Riegel in Ziemssen's "Handbuch." The latest and most complete collection of the literature is found in the monograph by West on "Plastic Bronchitis" [and the paper by Bettmann in the "Am. Jour. of Med Sciences," Feb., 1902. Bettmann gives all the references since Lebert's article in 1869. It is the most complete paper on the subject, of recent date.—Ed.]. Here follow the later communications referred to by me:

LITERATURE.

1877. Bernouilli: "Deutsches Archiv für klin. Medicin," Bd. xx, S. 363.
1878. Fraenkel: "Charité-Annalen," Bd. v.
— Degener: Schmidt's "Jahrbücher," Bd clxxix, S. 168.
1881. Pramberger: "Fibrinöse Bronchitis," Graz.
1882. Jäger: "Klin. Centralblatt."
— Mader: "Wiener med. Wochenschr.," Nr. 11 ff.
1883. Wolf: Dissertation, Würzburg.
— Escherich: "Deutsche med. Wochenschr.," Nr. 8.
— Vierordt: "Berliner klin. Wochenschr.," Nr. 29.
— Adserson: "Virchow-Hirsch's Jahresbericht," ii, S. 643.
1884. Möller: "Schmidt's Jahrbücher," Bd. cciv, S. 162.
1885: Mazzotti. "Klin. Centralblatt," S. 264.
1886. Sax: "Klin. Centralblatt," S. 614.
— Stark: "Zur Casuistik der Bronchitis fibrinosa," "Berliner klin. Wochenschr.," 1886, S. 221.
1889. Kisch: "Wiener med. Presse," Nr. 33.

1890. Model: "Ueber Bronchitis fibrinosa," Dissertation Freiburg.
1891. Roque: "Klin. Centralblatt," S. 489.
1892. R. Koch: "Petersburger med. Wochenschr.," S. 83.
— Hampeln: Ebenda, S. 336.
1893. Fritzsche: "Schmidt's Jahrbücher," Bd. ccxxxvii, S. 219.
— Beschorner: "Ueber chronische, essentielle, fibrinöse Bronchitis," "Volkmann's Sammlung," Nr. 73.
— Edgreen: "Klin. Centralblatt," S. 662.
— Duteuil: ibidem.
1894. Fedoroff: Cited in "Gazette des hôp."
1895. Magniaux: "Recherches sur la bronchite membraneuse primitive," Paris.
1900. Ott: "Fibrinous Bronchitis," "Münch. Med. Woch.," July 10.
1901. E. R. Le Count: "Tracheobronchial Fibrinous Cast," "Trans. Chicago Path. Soc.," vol. v, Oct. 14.
1902. Bettmann: "Fibrinous Bronchitis," "Amer. Journ. Med. Sci.," Feb.

Membranous and fibrinous exudates are formed in the bronchi under certain circumstances, and, when expelled by coughing, are found to represent casts of the bronchi. The remarkable appearance of these casts attracted the attention of physicians in the oldest times. At present several varieties of fibrinous bronchitis are distinguished, the most familiar being the *diphtheritic* and the *pneumonic* forms. Closely allied to the latter are certain other forms observed in the acute infectious diseases, and characterized by the fact that extension of the morbid process to the bronchial mucous membrane always takes place by continuity, either descending from the larynx or ascending from the alveoli. *Primary* or *idiopathic* fibrinous bronchitis has, however, been observed in a few instances.

The difficulty of separating this from the other, secondary forms is at times very great. Even when laryngeal croup spreads downward and bronchial coagula are expectorated, the laryngeal symptoms are so tremendous that the bronchi are practically forgotten, although the patient's condition, aside from the laryngeal affection, is most serious, and would probably terminate fatally.

Etiology.—The cases in which fibrinous coagula, either ribbon-shaped or of more or less cylindric form, are expectorated, may be divided into the following croups:

1. In infectious diseases, among which may be named: diphtheria, measles (Jäger), tuberculosis, variola, erysipelas, scarlatina (Adserson, Möller), typhoid fever (Eisenlohr, Möller, Svuque, Mazzontti), bronchitis, articular rheumatism (Degler), pneumonia (R. Koch) [influenza (Roque).—Ed.].
2. Fibrinous bronchitis in diseases of the lungs and of the heart.
3. Fibrinous bronchitis due to the effect of noxious fluids and vapors on the surface of the mucous membranes.
4. A toxic form after taking iodin (Fritzsche).
5. A so-called primary or idiopathic form; that is, one in which the etiology is not known.

In the cases which have been observed during or after infectious diseases, it is a question whether the presence of the infecting germ on the mucous membrane of the bronchi is to be regarded as the cause of the disease, or whether some other factor must be considered.

We know positively of two bacilli that may act as the primary cause of fibrinous bronchitis.

1. Primary fibrinous bronchitis is known to be caused by the *pneumococcus;* at least it presents the clinical picture of a true fibrinous bronchitis. The pneumococcus has, it is true, been demonstrated in many cases of simple purulent bronchitis, so that its etiologic significance in the production of fibrinous exudates is not altogether clear; but it is fair to assume that in cases in which it is the principal or only micro-organism present the fibrinous form of bronchitis develops, while the purulent form occurs when the streptococcus or some other cocci predominate.

The observation published by Magniaux tells of an eleven-year-old boy who was attacked with a severe cough and came to the hospital, because he began to cough up shreds of white membrane. He had violent attacks of dyspnea with cough, which were greatly relieved as soon as the membranes were expectorated. Gradually a mucopurulent expectoration developed, and signs of bronchopneumonic foci were found in the lungs. Then hemoptysis occurred several times, and bronchial membranes again appeared in the sputum, yet the condition constantly improved. Suddenly, however, symptoms of tuberculous meningitis appeared and the patient died. Cultures of Friedländer's pneumococcus were obtained from the membranes. No tubercle bacilli could be found in the sputa, although there was pulmonary tuberculosis in addition to tuberculosis of the meninges, peritoneum, and liver. Pneumococci were present in large numbers, and at times were found in pure culture in the discharge, while at other times they were mixed with other cocci and bacilli.

2. Primary fibrinous bronchitis may be caused by the *Löffler bacillus* (representing a true bronchial diphtheria), although this process is usually secondary.

[Councilman, Mallory, and Pearce,* in a study of 220 fatal cases of diphtheria, found in 42 a definite fibrinous membrane in the bronchi. Between this condition and bronchopneumonia there seems to be a definite relation; thus, in 100 cases with membrane in the lower respiratory passages (larynx, trachea, or bronchi) bronchopneumonia was present in 72%, while in the remaining 120 cases it was present in only 48%.

McCullom † has described complete fibrinous casts of the trachea and primary bronchi, which were expelled during a paroxysm of coughing by patients suffering from diphtheria (gives good illustration—photograph).—Ed.]

3. Picchini (cited by Magniaux and Bettmann) observed the disease in three workmen who had worked in a sewer. He could isolate three kinds of bacilli from the sputum, and one of these forms caused hemorrhagic inflammation on the trachea of a rabbit. In the case of

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* *Jour. Boston Society Med. Sciences,* vol. v, No. 5, 1900.

† "Medical and Surgical Reports of the Boston City Hospital," 9th series, 1898.

Robert Koch a pneumonia certainly occurred, but the pneumococcus was not to be found. We must assume that, besides the two forms mentioned, other micro-organisms may occasionally give rise to fibrinous bronchitis. [Strauss has found pneumococci, Staphylococcus pyogenes aureus and albus, Löffler's bacillus, and Streptococcus pyogenes.—Ed.]

The fibrinous exudations should not be confused with the fibrinous masses which are sometimes coughed up after hemoptysis, and which evidently represent the remains of true blood-clots. Such a case was demonstrated by S. B. West.* However, as a general rule, this does not occur as a result of the hemoptysis alone, some special factor being responsible for the production of these tube-like structures.

In Fraentzel's case of a consumptive soldier who had a pulmonary hemorrhage, the expectorated blood separated into two layers; in the upper layer were found tubular bronchial casts without cells or crystals and filled with blood. The symptoms improved, but on the eleventh day, with fever and the signs of endocarditis and nephritis, a similar discharge of blood occurred and resulted in death. On section, the mucous membrane of the bronchi was found not materially altered.

Hemorrhagic fibrinous bronchitis has, therefore, been described. The clinical course gives the impression of a malignant primary infection of the bronchi. The same is to be said of Kretschy's case, which requires further consideration.

Another independent form is that which occurs in pemphigus, unless we are willing to group it with that which occurs in variola and other infectious diseases.

Next follow the cases of the second group, which occur occasionally in affections of the heart. Hampeln demonstrated fibrinous exudates from bronchi of the second and third order, which such a patient has expectorated after puncture that was first followed by the usual serous discharge. Compare also Escherich's case in the bibliography. [Bettmann analyzed ten cases.—Ed.]

A special group is formed by the membranous exudates occurring as the result of *chemical influences*. Scalding with hot vapor may produce such an exudate (Parker). As is well known, experimenters have succeeded in producing croupous membranes in the trachea and larynx of animals by instilling ammonia. We also have the medicolegal reports of autopsies on human subjects killed by the inhalation of the vapors of ammonia. Here also a membranous exudate could be seen in the nasal cavity, in the throat, in the larynx, in the trachea, and continuing into the finest bronchi. But I know of no case in which the expectoration of such membranes has been reported. Recently observations have been published showing that tuberculous patients, after a protracted and liberal course of local treatment with lactic acid for tuberculosis of the larynx, sometimes have attacks of coughing in which they expectorate ribbon-shaped shreds of mucus from the trachea and bronchi.* In Fritzsche's case characteristic coagula of this kind were evidently due to the use of potassium iodid. Additional interest attaches to this case from the fact that it occurred in a patient with Basedow's disease.

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* *Brit. Med. Jour.*, Feb., 1880, p. 282; and Peacock, *Trans. Path. Society of London*, vol. XXIV, p. 20.

There still remain the so-called *primary or idiopathic forms*, in which we do not know the etiology and find the bronchi attacked primarily. The number of these diminishes greatly when we take away those which rest on a tuberculous foundation. The question of classification often has to be decided arbitrarily, for a case after scarlatina is not necessarily a case on account of scarlatina. On the other hand, no one will doubt that cases of very different etiology, according to our present knowledge, may present exactly the same picture. From all of which it appears that for some time it will not be possible to place this disease on a firm etiologic foundation.

[Bettmann's collection of cases was subdivided by him into nine groups, as follows:

I. Chronic bronchitis with expectoration of branching casts of the bronchial tree: twenty-seven cases.

II. Acute bronchitis with expectoration of branching casts of the bronchial tree: fifteen cases.

III. Cases in which branching casts were not expectorated, but were found in the bronchi at autopsy: six cases.

IV. Cases in which the casts expectorated showed dichotomous branching: eleven cases.

V. Expectoration of branching casts in organic heart disease: ten cases.

VI. Expectoration of branching casts in pulmonary tuberculosis: fourteen cases.

VII. Expectoration of small casts, often non-branching, in association with asthma: five cases.

VIII. Formation of casts in the bronchi in association with pulmonary edema following thoracentesis: four cases.

IX. Cases of doubtful classification because of incomplete reports: six cases.—Ed.]

We still therefore define fibrinous bronchitis as a disease which attacks the bronchi primarily and is characterized by dyspnea, occurring more or less frequently and terminating with the expulsion of fibrinous bronchial casts, to the great relief of the patient. It is exceedingly important to establish the etiology in individual cases; in practice it is of the greatest value to determine whether the case is one of true diphtheria or not. Many will wish to rubricate this form under an especial name because of the great practical importance of differentiation with respect to prognosis, treatment, and prophylaxis. It may even be asserted that, with the help of a bacteriologic examination, this differentiation can almost always be made with ease and certainty, but it will not be possible by the ordinary, older clinical methods of observation.

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* *Petersburger med. Wochenschr.*, XVII, p. 145, "Sitzung der Gesellschaft praktischer Aerzte zu Riga," Feb. 19, 1892.

Pathologic Anatomy.—The bronchial casts are cylindric structures: cylindric tubes in the larger bronchi, and small, solid threads in the smaller ones; or, vice versa, solid cylinders in the larger bronchi, and hollow, tubular casts in the smaller ones. Besides these two common forms, there are also rare cases in which the entire cast is solid or canalized, or, finally, the two conditions alternate. Often one may find inclosed in such a cast one or many small air-bubbles, like a string of beads. The ramifications of the cast correspond exactly to those of the bronchi; small tree-like casts have been observed 10 to 12 cm. (4 to 5 in.) in length. The extremities of the smallest finest twigs may be club-shaped, or they may be entirely smooth, like pack thread.

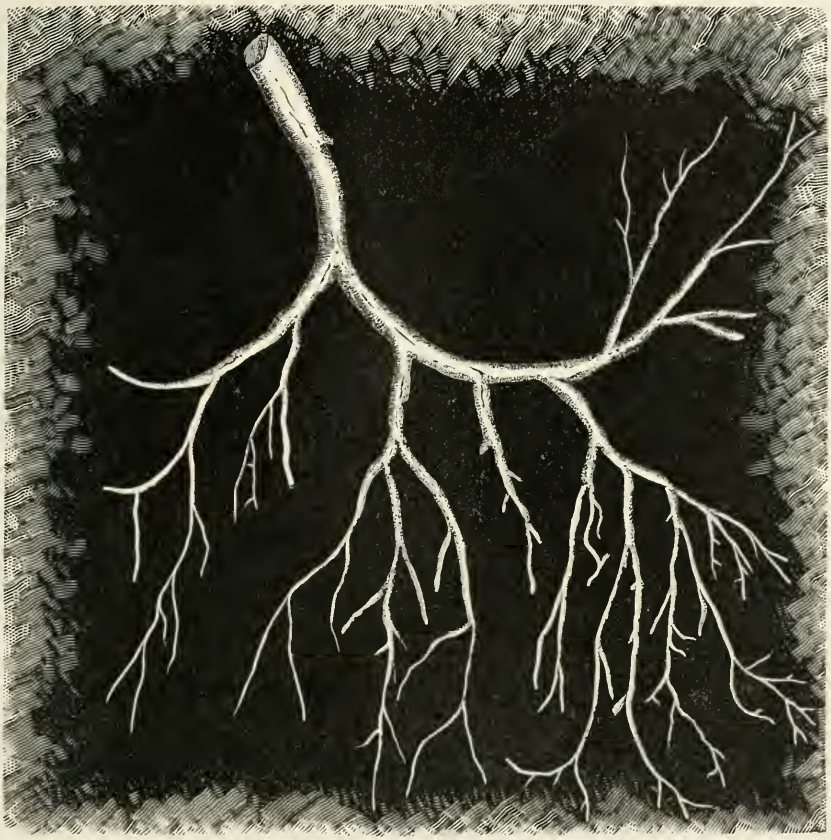

Fig. 5.—Bronchial cast expectorated during an attack of acute fibrinous bronchitis.

The *microscopic examination* shows that the casts have a laminated arrangement. In the center is seen a folded formation such as must develop when the exudate is produced from the mucous membrane in successive deposits, and new layers are constantly forming, while the older ones are pushed toward the free surface. In the central, oldest portion are found mostly * rudiments of cells, desquamated alveolar and bronchial epithelium, bacteria, and leucocytes [mononuclear (Roussell *et al.*, see Bettmann).—ED.], which, however, stain feebly. Here also are found Leyden's crystals, when they are present.

Robert Koch, who made a drawing of them in his case, represents the cross-section as hexagonal, which very much surprises me.† It is still more uncommon for the sputum to contain spirals, as observed by Friedreich, Zucker, Riegel, and Escherich. Flint even found hematoidin crystals. Fritzsche demonstrated in his case eosinophile cells, which were also increased in the blood.

Almost all authors agree that the masses expelled consist of fibrin; Fritzsche has also used the Weigert method of staining; Beschorner, however, declared that the ground-substance in his case was mucus. The casts are soluble in lime-water, and still more easily in the stronger alkalies.

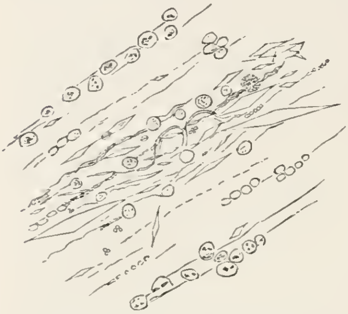
FIG. 6.—Longitudinal section from a bronchial cast hardened in alcohol.

The rapidity with which these casts may be formed is very surprising. It is most clearly shown in the valuable case-history contributed by Kretschy that they cannot be the result of cell activity or cell metamorphosis, but must be an exudate emanating directly from the blood- or lymph-vessels, which finds on the surface of the mucous membranes the conditions necessary for coagulation. Three large plugs were expelled in one day. In this case it is certain that the bronchi in which the exudate developed no longer possessed any epithelium.

[Bettmann found a surprisingly small portion of the sections take Weigert's fibrin stain. The greater number of fibrin fibrils are distributed in the outer layer or "skin" of the cast, or in the outer layer of the smaller whorls. The stain brings out organisms, streptococci and staphylococci, generally adhering to the outer side of the cast, though a very few are occasionally found in the mucus in the inside of the cast.

Hematoxylin and eosin: This stain shows a ground-substance consisting of two apparently distinct materials: a fibrillar substance taking a deep eosin stain, which is concentrically arranged about the lamina of the separate whorls. This incloses a second substance, which is transparent and takes a light tinge of hematoxylin. Undoubtedly this latter substance, from its optical properties and from its staining reaction, is mucin. The substance taking the eosin stain so deeply is not altogether fibrin, but in part shows itself to be such, for it contains the fibrillæ which take the fibrin stain. What the nature of the portions that do not take the fibrin stain is, it is difficult to say from the data at hand. Cells are very numerous, and are for the most part mononuclear leucocytes, though some polynuclears are seen. They are more numerous in the mucin ground-substance than in the eosinophilic material. There are a few alveolar cells, many of which contain blood pigment; a very occasional red blood-cell is seen. Besides these cellular elements, there are certain bodies which we supposed at first were cellular degenerations, but which in other stains took on a more specific character, and are, perhaps, some peculiar form of organism. There were also a small number of cells showing eosinophilic granules. None of these are polymorphonuclear leucocytes, but are either degenerating epithelial cells or large mononuclear leucocytes.

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* In some cases, no cell inclusions are found, as in that of Tuckwell, *Trans. Path. Soc.*, Bd. xxi.

† These crystals are discussed in detail in the section on Asthma.

Sections stained in eosin and methylene-blue: These show the ground-substance to be reticulated and taking altogether a deep eosin stain. The methylene-blue stains only the nuclei of the cells. There are no mastzellen. This stain brings out well the few eosinophilic cells.

The tubercle stain showed no tubercle bacilli, but brought out those peculiar bodies which are mentioned above. They are irregularly round, from 7 to 15 μ in diameter. (See Fig. 3, page 33.) They retain the fuchsin, keeping a deep red color. They have apparently a double contoured outer shell, from which in certain places the deeply staining protoplasm seems to have shrunk away. Vacuoles of various sizes are seen in their protoplasm. The protoplasm stains diffusely, but shows a few more deeply staining granules. The shell seems to have a tendency to roll up on itself.—Ed.]

In individual cases—as, for instance, in that of Waldenburg,* and especially in the two cases of Model—an astonishing amount of fat, both in the form of minute dust-like droplets and larger masses, was found between the fibrin fibers or in the fibrin flakes; large amounts of fat were also present free in the sputum and especially floating upon it.

In a few cases, as in the fifth of Model, lamination of the cast is not found. The seventh case of Model, in which the abundance of fat in the sputum suggested the thought of chylorrhea or lymphorrhea, presented macroscopically three distinct portions: One appearing milk-white, another bright red in color, and the third more gelatinous. The milk-white and rose-red portions contained the dendritic ramifications.

It is fair to assume that the exudates in the different pathologic conditions will be different, but it is not known what is especially characteristic of each variety; fibrin is no doubt found equally in the pneumonic, diphtheritic, and idiopathic forms. The pneumonic casts are generally brownish or reddish in color and contain large numbers of leucocytes; the diphtheritic are generally white and contain numerous remains of desquamated epithelial cells; while the idiopathic are also pure white. According to Lucas-Championnière's description,* some are found in which fibrin largely predominates, in others mucus, and in still others masses of fat; but the characteristic constituent in all is fibrin.

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* *Berlin. klin. Wochenschr.*, 1869.

[Bettmann states that Caussander concluded the casts were composed of syntonin.

From the review of the recent literature it seems probable that either mucin or fibrin may form the bulk of the coagula which characterize that form of bronchitis known as fibrinous, pseudomembranous, or plastic bronchitis.

The following authors have concluded from their investigations that the casts were chiefly, if not entirely, composed of fibrin:

Hint's † case showed fibrin coagula.

Strauss ‡ reports a case of chronic fibrinous bronchitis, and in the coagula were found many Charcot-Leyden crystals. The casts were made up mostly of fibrin. Strauss thinks that both mucin and fibrin coagula may exist and give rise to the same phenomena.

All modern writers, Brannan states, agree that the exudate is made up mainly of fibrin, containing in varying proportions epithelial cells, leucocytes, fatty matter, air-bubbles, and granular detritus. "According to Klebs (cited by Beschorner, *l. c.*), the casts are formed from transuded plasma of the blood, combined with an exudate of white blood-cells which have escaped from the vessels."

Herzog § examined the coagula of two cases of true fibrinous bronchitis, and found that fibrin made up the bulk of the casts.

Sokolowski ‖ reports four cases of idiopathic fibrinous bronchitis in which the casts were made up of fibrin and white and red corpuscles.

Patton and Herzog ** report a case in which the bulk of the mass of the exudate showed a fibrillar arrangement, and stained blue, giving a typical fibrin stain. The casts consisted of fibrin mixed with swollen degenerating epithelial cells, and a considerable number of leucocytes.

Vintras †† reports a case of plastic bronchitis in which the coagula were found to be made up mainly of fibrin, with some leucocytes, and epithelial cells. Charcot crystals were absent.

Lépine ‡‡ reports a case of chronic pseudomembranous bronchitis, in which coagula composed of fibrin and epithelial cells were expectorated.

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* Thèse de Paris, 1876.

† *Wiener med. Wochenschr.*, 1898, XLVIII, 1991–1997.

‡ *Berlin. klin. Wochenschr.*, 1900, No. 19, p. 407.

§ *Centralbl. f. allgem. Path. u. path. Anat.*, 1898, VIII, 1008–1010.

‖ *Deutsches Archiv. f. klin. Med.*, 1895, 1896, LVI, 476–489.

** *Jour. Am. Med. Assoc.*, Jan. 1, 1898, p. 25.

†† *Lancet*, Sept. 15, 1900.

‡‡ *Revue de Méd.*, Paris, 1898, XVIII, 835–838.

Nachod* and Fritsche† report cases in which the casts were undoubtedly made up of fibrin.

Schittenhelm's ‡ two cases were carefully investigated, and both color and chemical tests applied. The coagula in both cases were found to be made up mainly of fibrin. The casts were made up of lamellæ, of fine fibers, which in places made a network inclosing epithelial cells and leucocytes.

Huchard and Claisse's § case also gave fibrin in the casts.

Schmidt ‖ reports a case in which he chemically investigated the nature of the casts, and found them to be made up mostly of fibrin. Abundant, desquamated, fatty alveolar epithelium was also found, as well as hyaline blood-cylinders, "*corpora lecithinoidea*," and "lecithinoid cylinders."

Eppinger's** investigations with chemical and color tests go to show that the coagula are formed of fibrin.

On the other hand, other observers come to different conclusions:

Thus, Beschorner †† reports two cases of fibrinous bronchitis, in which the cases were examined by Neelsen. The latter found the bulk of the coagula to be made up of thickened mucus, with only a few shreds of fibrin.

Klein ‡‡ says that, in a case of fibrinous bronchitis observed by him, the expectorated mass was made up of mucin, which did not take Weigert's fibrin stain.

Habel §§ reports 3 cases of chronic fibrinous bronchitis in which the coagula were formed of mucin. The reaction of the casts was acid. As mucin coagulates in the presence of acid, Habel explains the formation of the casts by the coagulation of the mucus in the bronchi, due to the presence of acid, most probably formed by the action of bacteria.

Grandy ‖‖ speaks of a case of so-called chronic bronchial croup in which the expectorated masses were made up of mucus, with white and red blood-corpuscles, and some bronchial epithelial cells, many of which were swollen and filled with mucus. Grandy expresses the opinion that a true fibrinous inflammation of the bronchi may take place, and run a similar course.

Brannan *** quotes Beschorner.

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* *Prager med. Wochenschr.*, 1897, Nos. 3, 5, and 6.

† "Schmidt's Jahrbücher," 1893, No. 237, p. 138.

‡ *Deutsches Arch. f. klin. Med.*, LXVII, vols. 3 and 4, p. 336.

§ *Journal des Praticiens*, 1895, p. 117.

‖ *Centralbl. f. allgem. Pathol. u. path. Anat.*, June 15, 1899.

** "Ergebnisse der allgemeinen Pathologie u. pathologischen Anatomie," Part 3, p. 200.

†† "Volkmann's Sammlung klin. Vorträge," 1893, No. 73, p. 640.

‡‡ *Wiener klin. Wochenschr.*, July 30, 1896, p. 710.

§§ *Centralblatt f. innere Med.*, 1898, XIX, 9–12.

‖‖ *Centralbl. f. allgem. Path. u. path. Anat.*, Bd. VIII, 1897, p. 513.

*** *Med. News*, 1896, LXIX, 169–170.

We find, again, that the structure of these casts is determined by Wagner and others to result from cellular changes:

Thus, E. Wagner (cited by Beschorner, *l. c.*) believes that the casts owe their origin to a peculiar metamorphosis of the epithelium of the bronchial mucous membrane, by which the epithelium forms the cellular elements by endogenous cell production and the fibrinous framework out of the remaining cellular substance.

Still another conclusion is arrived at by Lehmann-Model,* who explains the formation of the coagula by assuming the existence of a chylous exudation, but his views have not been accepted.—Ed.]

The question in regard to the condition of the bronchi is not yet entirely settled. From the experiments of Weigert and the findings of Kretschy it is assumed that the epithelium of the bronchi must be destroyed and cast off before the formation of a fibrinous exudate becomes possible. Good observations on the condition of the bronchial epithelium have rarely been made, evidently on account of the practical difficulties, while the casts have often been examined by the best methods. [Schittenhelm † found a desquamative catarrh of the alveoli and an exudation into the bronchi, bronchioles, and alveoli (Bettmann).—Ed.]

Course.—The idiopathic form of bronchitis fibrinosa is usually a chronic disease. [Bettmann collected twenty-seven cases.—Ed.] After the patient has suffered several times from bronchitis, attacks occur in which, with violent fits of coughing and dyspnea, masses are expelled. These, if very carefully disentangled in water, present themselves as casts of a portion of the bronchial tree. We can easily recognize the characteristic branching. Usually such casts are formed only in a certain portion of the bronchi; the patient has the sensation that his breathing is becoming more difficult; he feels a sense of pressure or even a humming, which can be felt on the thoracic wall, and finally, after the expulsion of the exudate, these symptoms disappear and the patient experiences the greatest relief. Then the play begins again. In this way casts may be expectorated several times during the day; soon, however, the attacks become more infrequent, and finally stop altogether if no accidental injury causes an exacerbation. The disease may drag on in this way for months or years. The case of Kisch lasted for over twenty-five years.

In many cases the symptoms are so mild that they scarcely annoy the patient, while in others they are very violent. The attacks follow one another rapidly, fever develops, and in half of the cases the patient dies during an attack; the duration varies between two and fourteen days. This rare form of the disease has been designated acute fibrinous bronchitis. [Fifteen cases (Bettmann).—Ed.]

So, for instance, the case of Jäger was that of a child who, after measles, was attacked with catarrhal symptoms and dyspnea; tubular membranes were expectorated, and on the fifth day the patient died. There was a croupous membrane, very slight in the larynx and becoming more and more marked in the deeper structures; it had filled the bronchial branches of the left lung.

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* Inaug. Diss., Freiburg, 1890, "Ueber Bronchitis Fibrinosa."

† *Deutsch. Arch. f. klin. Med.*, S. 340.

Especially valuable is the very exact observation of Kretschy. He reports the case of a twenty-three-year-old shoemaker, who was quite suddenly attacked with chill and on the next morning expectorated a reddish mass. The fever was observed from the third day.

	Temperature.	Pulse.	Respiration.
10	38.0° C.—100.4° F.	68	36
	38.6° " —101.4° "	92	32
11	37.7° " — 99.8° "	88	32
	41.0° " —105.8° "	128	44
12	37.4° " — 99.3° "	88	32
	40.4° " —104.7° "	120	36
13	39.0° " —102.2° "	100	34
	40.9° " —105.6° "	148	60–72
14			
15	39.4° " —102.9° "	128	48
	40.3° " —104.5° "	120	72
16	39.8° " —103.6° "	128	72
	40.2° " —104.3° "	120	64
17	40.0° " —104.0° "	140	60
		164	78

The membranes were formed in the middle and lower lobes of the right lung.

That menstruation and pregnancy sometimes have an influence on the course of the disease cannot be considered strange.* [In Schnittzler's case attacks replaced menstruation for many months after the sixth, seventh, and eighth pregnancies.—Ed.]

Between the acute and chronic forms there are some which represent transition forms and which have been distinguished as subacute cases. To this class belongs a case of Escherich, characterized by a febrile movement which on two occasions immediately preceded the beginning of menstruation and ended by crisis after a duration of eight days. This was followed by a longer afebrile stage with bronchitic symptoms, which gradually led to recovery.

Symptoms.—*Sputum.*—The characteristic features are the coagula which have already been described. Aside from these, the sputum is catarrhal in character. On standing in the air it often takes on a greenish color. This has also been frequently observed in other kinds of sputum, especially the purulent. *Blood* is also quite frequently present, and may be so abundant as to constitute a true hemoptysis. This hemoptysis [occurring in one-third of the cases.—Ed.] should not by any means be regarded as a sign of tuberculosis; it is not the cause, but the result of the fibrinous bronchitis. We can readily understand that when coagula are torn away from the bronchial wall by the violent fit of coughing, blood-vessels in this wall may easily be injured to a considerable extent. Other noteworthy observations have at times been made in the investigation of the sputum. *Asthma crystals* and *asthma spirals* have been found; the former more frequently than the latter.

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* Oppolzer and Brik, *Wiener med. Presse*, 1882, p. 828, 861.

Dyspnea is very marked in most cases. The attacks are connected with the expectoration of the coagula and subside when the latter are expelled, to return at certain intervals, the duration of which naturally depends on the intensity of the coagulative process, the extent of the false membrane, and the sensitiveness of the patient. In acute cases the dyspnea is usually continuous, while in chronic cases the intervals between the attacks are usually quite free from this symptom; but the attack is generally preceded by a period in which the shortness of breath gradually increases. Besides these, we find a number of cases described in the literature in which the patients suffer very little dyspnea, and some in which there is none at all; the membranes are discharged without any effort or difficulty and the general health is quite good. The albuminuria which occasionally occurs in the more severe attacks of dyspnea can now, according to the results of Schreiber's experiments, be explained as a mere transient consequence of the circulatory disturbance.

Cough accompanies the dyspneic attacks and the expectoration. It may succeed very easily in expelling large masses of sputum; it may be laborious, distressing, and bring up very little discharge; all possible grades have been observed. The cough is at times accompanied by a peculiar flapping or purring sound ("Flattergeräusch").

Auscultation.—The variable signs observed on auscultation are easily explained by the behavior of the false membrane. Thus, the breath sounds may be greatly weakened or only slightly diminished. Muffled, moist râles of every form and size may be heard. Occasionally a fluttering or flapping sound is produced by the movement of the partially separated membrane, which may even cause a palpable thrill of the thoracic wall.

Percussion gives nothing characteristic, if there are no complicating diseases of the lungs.

Inspection: The patients are often somewhat cyanotic, sometimes extremely so. At the height of the dyspnea the affected lung lags in respiration.

Fever belongs to the acute form, and nothing characteristic is yet known in regard to its course.

Splenic enlargement has very frequently been noticed.

Hydropic conditions and *albuminuria* occur only exceptionally as complications.

The general condition may be quite variable, as can be readily inferred from the changeable symptoms. [Loss of flesh and strength are not uncommon.—Ed.] From patients who suffer most severely to those who are practically well, all grades may be observed.

Complications.—Tuberculosis, pneumonia, and bronchitis are the most frequent. Model lays the greatest stress on tuberculosis because he found it ten times in 21 autopsies; he believes, therefore, that not only does tuberculosis produce a certain predisposition to fibrinous bronchitis, but that there is a more intimate relation between the two diseases. A great variety of other lung and heart diseases have also been observed. Much that was mentioned in the etiology might from another standpoint have been discussed here. I will name only the extensive ulcerations of the trachea and bronchi (Gumvens, after Biermer).

Complications with skin eruptions are not frequent, but have attracted considerable attention: Waldenburg,* impetigo capitis; Streets,† herpes zoster and impetigo; pemphigus has been given by Mader as a direct etiologic factor.

Diagnosis.—The greatest difficulty may arise in differentiating this disease from bronchial asthma. Vierordt observed a thirty-one-year-old mountaineer who had for years suffered from expiratory dyspneic attacks. In the abundant sputum were found gray flakes, and thick, grayish-white bronchial casts; Leyden's crystals were always found, but only once was a Curschmann's spiral observed.

Andral considers the diagnosis assured whenever, in the course of a simple bronchitis, a dyspneic attack occurs and the respiratory murmur is no longer heard over a certain area of the lungs, while the percussion note is unimpaired. But even in such cases a diligent search for the characteristic sputum should be made.

The etiology must be determined and the existence of diphtheria established or excluded; this usually requires a bacteriologic examination. It is well known that chronicity has been observed in diphtheria, and this may confuse the diagnosis, and it is certain that there is no such thing as a diphtheritic form of acute fibrinous bronchitis. Pneumonia is also to be thought of. Cases which are the result of a local injury or of the use of drugs are easily excluded.

Treatment.—The treatment will depend upon the cause, so far as we can find this out. In the acute cases Biermer advises active mercurial treatment; in all acute, severe inflammations upon the nature of which a certain amount of doubt rests this is still, as formerly, highly recommended by those physicians who adhere to the heroic methods. The inhalation of warm water vapor unquestionably affords relief to the patient, or lime-water may be nebulized and inhaled. Lime-water undoubtedly exercises a certain dissolving influence on the false membrane. In addition to these remedies, potassium iodid, creosote, turpentine, and tar (internally) have been highly lauded in individual cases. [The first-named by English and American practitioners.—Ed.]

In general, the management of the disease should be based on the principles laid down for the treatment of simple bronchitis. Nutritious diet and careful nursing are the more important, as the greater number of these cases rest on a tuberculous foundation. Whether, under such circumstances, creosote is of preeminent advantage is a question which requires further investigation.

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* *Berliner klin. Wochenschr.*, 1869.

† "Schmidt's Jahrbuch," Bd. CLXXXVIII, p. 254.

PUTRID BRONCHITIS.

Older literature, history, and detailed description by Lebert, "Klinik der Brustkrankheiten," I, p. 102, 1874. All clinical treatises on bronchiectasis include putrid bronchitis. It is also treated in connection with gangrene of the lungs.

LITERATURE.

Traube: "Deutsche Klinik," 1853, 1861, 1862.—Empis: "Catarrhe pseudogangreneux," "Gaz. des hôp.," 1863, p. 253.—Gamgee: "Edinburgh Med. Jour.," Mar., 1865, I, S. 807, 1124, "Buttersäure und Milchsäure bei Gangrän der Lunge nachgewiesen."—Leyden und Jaffé: "Deutsches Arch. für klin. Med.," Bd. II, S. 488, 1867.—Rosenstein: "Berlin. klin. Wochenschr.," 1867.—Loos: "Ueber putride Bronchitis," ibidem.—Lancereaux: "Archives de méd.," 1873, "Lungengangrän."—Gerhardt: "Deutsches Arch. für klin. Med.," XV, 1875.—Leyden: "Virchow's Archiv," Bd. LXXIV, S. 414, 1878. (Tyrosin crystals.)—Kannenberg: "Charité-Annalen," V, 1878.—Leviez: "De la bronchite fétide," Thèse de Paris, 1883.—Lacher: "Münch. med. Wochenschr.," 1887, Nr. 33.—Lumniczer: "Ref. klin. Centralblatt," 1889, S. 51.—Lancereaux: "Clinique méd. de la Pitié," 3^{e} sér., 1890.—Rendu: "Clinique méd.," 1890.—Loebisch und Rokitansky: "Centralbl. für klin. Med.," 1890, Nr. 1.—Köhler und Bardeleben: "Berlin. klin. Wochenschr.," 1891, 9. Februar.—Bernabei: "Ref. klin. Centralblatt," 1894, S. 979.—Hitzig: "Beiträge zur Aetiologie der putriden Bronchitis," "Virchow's Archiv," Bd. CXLI, S. 28, 1895.—Laycock: "On Fetid Bronchitis," "Edinb. Med. Jour.," p. 961, 1865.

Etiology.—We have to deal not so much with a definite nosologic entity, as with a peculiar form of decomposition of the sputum, which may occur in any disease of the lungs or bronchi. As soon as this decomposition occurs, the disease assumes a special character and the consideration of putrid decomposition of the sputum under a separate heading is thus justified. But it is not an independent disease, and while the name "putrid bronchitis" is very convenient, it should be used with this reservation. Bernabei, on the strength of his bacteriologic investigations, claims recognition for a true primary putrid bronchitis.

Marfan, in the "Traité de Médecine," has introduced the name *gangrène des bronches*. He insists that there is gangrene of the bronchial mucous membrane; although all that has been shown is that the mucous membrane becomes involved whenever the contents of the bronchi undergo putrid decomposition; the new term, therefore, merely adds to the confusion, as it leads many to believe that an entirely new condition is meant. He suggests the question whether there is putrid bronchitis without gangrene of the bronchi, independent of bronchiectasis, occurring simply as a result of the infection of the bronchial contents. He does not deny this, but says that such a case is a mere passing complication, without danger; that the only fatal case is one reported by Sée, in which the autopsy showed not the slightest change in the bronchi or lungs.* Considering the quantity of air daily drawn through the bronchi, and how absolutely certain it is that this air contains all manner of lower organisms which occasionally penetrate into the smallest twigs of the bronchial tree, and even into the alveoli, there can be no doubt that some protective structures exist in the bronchi. Not only is the dust caught in the tough masses of mucus, and either carried out again or deposited in some safe place, but a similar fate awaits the micro-organisms that have eluded the intercepting mechanism in the nose, larynx, and trachea. Surrounded by mucus and taken up by the cells, they are either expelled or deposited in lymph-glands, furnishing a ready explanation of the well-known fact that the bronchial glands in perfectly healthy people are often found to contain tubercle bacilli.

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* It is found in *Gaz. med.*, 1881.

As long as all these structures functionate properly, there is no danger of the bronchial contents undergoing decomposition. Just as in the case of abnormal fermentation in the stomach or in the bladder, disease must first destroy the normal protective agencies before micro-organisms can effect an entrance and obtain the mastery by transforming the medium to suit their conditions of life. Decomposition is accordingly to be attributed to invasion of the bronchi by micro-organisms capable of producing fermentation, and to a diseased condition of the bronchi favoring the growth of such micro-organisms; hence it is found in bronchiectasis, with foreign bodies, in all suppurative processes of the lungs, after all the infectious diseases, especially typhoid fever, influenza, and pneumonia,* and no doubt also in simple bronchitis. When a traction diverticulum (of the esophagus) ruptures into a lung cavity, the phenomena of putrid bronchitis may appear, as shown by a case of Tiedemann.† The relation between putrid bronchitis and tuberculosis is rather one of mutual exclusion, since it is very rare to find the two diseases associated. The exact nature of the cause of the decomposition as yet remains unknown; the discharge, of course, contains large numbers of different kinds of cocci and other micro-organisms. But Bernabei and Lumniczertin some of their communications claim to have found a characteristic micro-parasite which they describe.

In a case reported by Rosenstein a young girl, after inhaling Oidium albicans, developed a putrid bronchitis. Canali described a case of putrid bronchitis following actinomycosis of the air-passages. These observations do not, however, justify the conclusion that Oidium albicans and actinomyces are the causes of putrid bronchitis, although they no doubt favored the growth of the germs that produced the decomposition.

J. Lumniczer ‡ isolated a number of micro-organisms from the putrid sputum; among them was a bacillus, an agar culture of which after six to seven days emitted the same odor as the putrid sputum itself. He regards this as the cause of putrid bronchitis. Bernabei appears to have found the same organism.

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* Sée doubts this, but is mistaken. † *Deutsches Arch. für klin. Med.*, vol. xv.

‡ *Klin. Centralblatt*, 1889, p. 51.

The most recent examination of the sputum of putrid bronchitis comes from Eichhorst's clinic (Hitzig). Two closely related bacilli were found, which were similar to the colon bacillus and, in bouillon culture, emitted a stale and often fetid odor.

[Leptothrices have been found in the fungoid plugs of putrid bronchitis by Leyden and Jaffé, Dittrich, and Traube; and in the tracheal secretion of a patient with pharyngomycosis and ozæna trachealis by Baginsky; Buchholtz found streptothrices in the case of a necrotic peribronchitis.—Ed.]

Numerous **chemical analyses** of putrid sputum have been made. The chief findings are decomposition products; thus, methylamin and butyric and acetic acids were found by Gregory and reported by Low. The same author found traces of butyric acid in every sputum examined. Jaffé found leucin and tyrosin in very small quantities, so that after simple drying of the sputum no recognizable crystals are to be expected. Löbisch and Rokitansky were able to demonstrate diamin. At times hydrogen sulphid and ammonia are present. Jaffé obtained from the so-called Dittrich's plugs a white, easily pulverized substance, which stained blue on the addition of iodin. Filehne and Stolnikow isolated a ferment which bore a certain resemblance to pancreatic ferment.

The similarity of this sputum to that of gangrene of the lungs, and the fact that putrid bronchitis often passes over into the latter, suggests the thought that the two diseases are very closely related; that the same process is at work in both cases, in the one affecting the alveoli, and in the other the bronchi. By putrid bronchitis we understand the form in which the changes in the bronchi and lungs do not extend deeper than in simple catarrh; and it is only the peculiarities of the sputum that justify us in discussing the condition separately.

Pathologic Anatomy.—The pathologic findings are very few. The bronchial wall is in a certain condition of softening, and denuded of its epithelium; the underlying tissue is covered with a semi-solid material. In places the subepithelial tissue is attacked and develops a gangrenous degeneration, which may extend deep into the lung tissue. This explains the occasional development of gangrene of the lungs from putrid bronchitis.

Symptoms.—1. The *sputum* has an extremely bad odor, resembling that in gangrene of the lungs—an odor which we perceive as soon as we approach the patient, and which is decidedly different from any other, even the most nauseating, odor from the mouth. The discharge is expelled without effort and in considerable quantities; it is not unusual for 300 to 400 c.c. (10 to 14 ounces) to be discharged in a single day.

When poured into a glass, the sputum separates into three layers: The uppermost is frothy, consisting of mucus, which floats on top because of the air contained in it, and which shows an upper portion especially rich in air-bubbles, from which large brownish flakes of mucus hang down into the second layer. In the first we often find well-preserved pus-cells and epithelial cells and also many that have undergone fatty degeneration. Free fat drops may be seen floating about.

The second layer is called the serous layer, as it consists of a thin fluid, which, however, is not serous; it is of a dirty, brownish or sometimes yellowish-green color; formed elements are not found in this layer.

The third layer is the sediment; in it are found large numbers of vibriones, spirilla, and cocci; detritus; fat drops; and peculiar, horribly offensive plugs which contain fat-needles (very beautiful, long, round, and gracefully curved), leptothrix, monads, and large numbers of very fine granules, derived from the degenerated cells; these, as they were first described by Dittrich, are known as *Dittrich's plugs*. They vary in size from the very small or almost microscopic ones to those of the size of a bean.

Not infrequently these plugs contain no cells at all or only a few. (Are these younger, and the former the older plugs?) The leptothrix threads may be stained a beautiful violet or blue color with tincture of iodin. At times pigmented granules and the remains of red corpuscles are seen. Hitzig claims to have found hematoidin crystals.

If the fatty acid needles are crowded together in groups and attain a considerable length, as is not uncommon, they may be mistaken by an inexperienced physician for elastic fibers; but they are dissolved by caustic alkalies, melt on the application of heat, are pointed at the end, and never branch; their recognition is therefore easy.

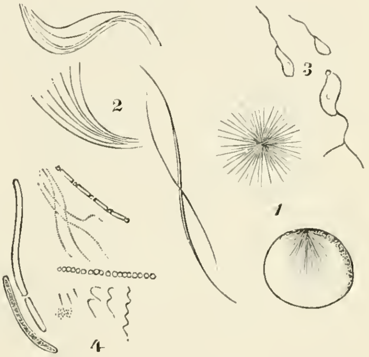

FIG. 7.—From the sediment of the sputum in putrid bronchitis: 1, Fatty crystals, free and within cells; 2, fatty acid needles; 3, monas and cercomonas; 4, leptothrix; other fungi; spirilla and bacteria.

Traube believes that the plugs at first consist chiefly of pus corpuscles and a finely granular detritus, which increases, while the cells disappear and fat droplets and fatty needles accumulate in greater and greater numbers in the plugs. He therefore constructs four stages,* which is, of course, artificial. We must confess that we do not know how these plugs are formed and how they develop; their foundation and chief constituent is a fungoid mass which has never been sufficiently studied, but is quite similar to leptothrix.

The monads, *monas* and *cercomonas*, described by Kannenberg have been more minutely studied by Streng, who found that they multiplied rapidly in bouillon at 37° F.

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* "Gesammelte Abhandlungen," II, S. 686.

2. *Cough* is characterized by its frequency and the ease with which large masses of sputum are expectorated.

3. *Fever* is a very frequent symptom, which has a very baneful influence on the prognosis. It shows that a considerable quantity of the decomposed material has passed into the circulation and that a septic infection is developing. Lighter grades of this infection may last for some time and yet the patient eventually recover.

Course.—In simple uncomplicated cases occurring in strong individuals, the putrid condition after some time—one to several weeks—usually disappears and recovery takes place. In such cases the disease has not infrequently a tendency to recur. In other cases a septic fever consumes the patient, or complications may occur which terminate life. Of these complications, some are explicable by the influence of the sputum on the walls of the bronchi and on the neighboring lung tissue; these are ulcerations and gangrene of the lungs, catarrhal pneumonia, and diffuse bronchitis. The others are metastatic in their nature: such are fetid abscesses in the various organs. One was seen by Lacher in the left occipital lobe.

Diagnosis.—Under this heading we must once more emphasize the fact that we are not dealing with an independent disease. The correct diagnosis must always bring into prominence the anatomic basis of the disease. Practically the disease which we have in mind is bronchiectasis with putrid decomposition of the bronchial secretion. The statistics of so-called putrid bronchitis are so generally collected from these cases that all others become insignificant. Pure bronchitis with putrid decomposition of the secretion is so rare a disease that I have found only the assertions of authors that there is such a disease, but no authentic history of a case. The diagnosis of putrid bronchitis owes its popularity to the convenience with which it can be made; in truth it is not a complete diagnosis; the pulmonary condition which has caused the putrid decomposition should always be determined. It is a matter of the greatest difficulty to decide whether the bronchial walls are in general intact or whether a putrid decomposition has penetrated into the lung tissue. Gangrene of the lungs is recognized by the great tendency to hemoptysis and, on microscopic examination of the sputum, by the presence of ragged shreds of lung tissue, which, however, show hardly a trace of the characteristic elastic fibers; for the elastic fibers are very quickly destroyed by the gangrenous fluid, and disappear before the fragment of tissue is expectorated. This detail is not as important from a clinical standpoint as might be supposed; the important factors in the diagnosis are the presence of fever and the general condition. Any case, even an apparently mild one, may rapidly become septic. *A priori*, it is natural to suppose when the lungs are sound there is less danger than when the lungs are affected; but a bronchiectatic subject is never safe, and in the present state of diagnosis every case must be regarded clinically as bronchiectatic if there is a putrid discharge and gangrene of the lung cannot be diagnosed. It is not possible to diagnosticate putrid bronchitis in the narrowest sense of the term.

If in a case of previously known pulmonary disease with chronic cough, indefinite dulness, and the absence of tubercle bacilli from the sputum, fetid expectoration develops, the diagnosis of bronchiectasis with putrid decomposition is very probably correct.

If a neighboring cavity, an empyema, an abscess of the vertebræ, of the liver, or of the lung itself, or an echinococcus sac ruptures into the bronchi, the sputum may assume a fetid character; and if this occurs suddenly, the existence of such a source must always be thought of.

If tubercle bacilli are found in putrid sputum, it may be assumed that the walls of a tuberculous cavity have become gangrenous, as in such cases (of tuberculosis) the process of decomposition is never limited to the bronchial contents.

Treatment.—In the treatment of the condition the disinfecting power of certain drugs, which are made to act on the bronchi as much as possible, is utilized. The oldest procedure which still enjoys great popularity is the inhalation of turpentine. A simple and very useful method consists in pouring the turpentine on hot water and having the patient draw deep breaths with his mouth directly over the vessel. A better way is to pour the turpentine into a flask like an ordinary atomizer, provided with a suitable mouthpiece on one side and a rubber bulb on the other; air is driven through the flask and, if the latter is placed in hot water, becomes charged with the fumes of turpentine. This medicated air is inhaled by the patient. More recently various forms of inhalers or masks have been devised in which a sponge is held under the patient's nose and mouth and turpentine or a solution of carbolic acid poured on the sponge drop by drop. With these masks a more protracted inhalation is possible than with an atomizer.

In addition, it is customary to administer internally one of the substances that are excreted by the bronchial mucous membrane and thus act as disinfectants. For this purpose also turpentine is the favorite remedy. Other substances, such as carbolic acid, tincture of eucalyptus, sodium hyposulphite (Lancereaux), and tincture of benzoin, have also been recommended. Eichhorst introduced myrtol, and found numerous imitators. Vigorous supportive measures to improve the general condition, nourishing food, cod-liver oil, plenty of exercise in the open air, are not to be neglected.

When by the help of these measures the putrid character of the sputa has been made to disappear, the treatment of the fundamental condition again asserts its rights.

TUBERCULOSIS OF THE BRONCHI.

Clinically tuberculosis of the bronchi is never regarded or treated as a separate disease; it is known only in connection with pulmonary tuberculosis, to which it is generally assumed to be secondary. But this theory is not to be accepted offhand. It is to be remembered that in the great majority of cases tuberculosis must be regarded as a disease which, through the invasion of the tubercle bacillus, attacks certain predisposed organs. It is undoubtedly true that the lungs of individuals who, either congenitally or because of their mode of life, possess a low grade of resistance are especially predisposed to the disease.

The manner in which the pulmonary infection takes place has never been clearly established. It is possible that tubercle bacilli are inhaled and find openings between the epithelial cells through which they are able to pass into the subepithelial tissue. It is possible that the bacilli penetrate as far as the alveoli and then follow the same path as soot and dust; that is, they are carried off by the lymph vessels and deposited in the bronchial glands or in the follicular apparatus of the bronchi. Here they establish themselves and cause enlargement and caseation of the glandular structures, which in their growth gradually encroach upon, and finally perforate,* the bronchial wall and pour out their infectious contents on the surface of the mucous membrane. This infectious material is then by aspiration drawn into various portions of the bronchial tree and leads to wide-spread infection of the pulmonary tissue.

Finally, there is the possibility that the inhaled bacilli may deposit themselves in the alveoli and there immediately cause the development of caseation.† Whether all these possibilities take place, and which of them takes place most frequently, has not, in the author's opinion, been decided by the works that have so far appeared on this subject. Some investigators even doubt that infection of the lung takes place only through inhalation, believing rather that the infectious agent is frequently introduced through the blood channels.

Pathologically two processes have been demonstrated in the bronchi: caseous bronchitis and the development of bronchial tubercles. Neither of these two is known to us clinically; they are marked by the complicated symptoms of phthisis. A primary, to say nothing of a pure, tuberculosis of the bronchi is clinically unknown as yet. Nevertheless I have no doubt at all that there is a primary tuberculosis of the bronchi, as well as a primary tuberculosis of the larynx or of the urinary passages. The tuberculosis of children is essentially a tuberculosis of the bronchi; it is, as we all know, characterized by disease of the bronchial glands, as evidenced by the presence of marked dulness between the shoulder-blades, and the fact that the cough is usually spasmodic and paroxysmal in character. It is further characterized by the rarity of cavity formation, of hemoptysis, of chronic circumscribed apical involvement, and, on the other hand, by a tendency to appear in the form of a lobular pneumonia, and to produce metastases in other organs, especially in the serous membranes. Bouchat uses the term "bronchial phthisis."

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* Michael, "Jahrbuch für Kinderkrankheiten," New Series, vol. XXII, p. 30, 1884.

† The possibility that not the bacillus alone, but some derivative, either an amorphous product of its activity, or morphologic elements that have in some way been irritated and predisposed by the bacillus, is responsible for the propagation of the disease need scarcely be taken into consideration in the present state of our knowledge. The theory cannot however be definitely discarded.

Tuberculous bronchitis shows an infiltration of the wall with a mass of small cells, producing considerable thickening of the wall. The bronchial walls are converted into a yellowish-white, homogeneous mass of small cells, which is quite sharply defined on the outer side and, at the lumen, is limited by a substance consisting of inspissated secretion and softened caseous material. The epithelium is destroyed, wherever it comes in contact with these masses, and the friable material is ultimately discharged into the bronchi. Here a caseous ulceration of the bronchial wall takes place. Caseous bronchitis arises mostly in the smallest bronchioles, where they open into the alveoli, and spreads from these to the larger branches.

Rindfleisch places the beginning at the point of transition between the smallest bronchi and the alveoli, which he declares is the regular starting-point of the disease; but I do not find this exclusive mode of origin confirmed by other authors. There are certainly cases in which the disease begins in the larger branches, and from these advances to the smaller ones.

The final stage of caseous bronchitis is ulceration, unless the disease becomes arrested and absorption takes place—possibilities that are always present, although they may not be fulfilled.

Caseous bronchitis also has a great tendency to penetrate more deeply and cause the development of peribronchial inflammation. This caseous peribronchitis represents the transitional stage between disease of the bronchi and disease of the lungs.

It follows, therefore, that caseous bronchitis has a definite seat, a positive starting-point, and a well-defined area of extension—namely, the subepithelial connective tissue of the bronchi, which represents a follicular tissue very rich in vessels (both blood- and lymph-vessels), and in a sense limited externally by the circular layer of smooth muscle-fibers. The latter, of course, do not arrest the disease; on the contrary, they always participate in the morbid process, which continues to advance and does not even respect the cartilage.

An effort has been made to draw a strict line of separation between tuberculous bronchitis and tuberculosis of the bronchi, and even now that the cause of both has been shown to be identical, we still adhere to the distinction. As a matter of fact, the development of circumscribed areas of true tubercle, as opposed to the diffuse infiltration of caseous inflammation, represents such a very different form of the disease that the distinction will no doubt continue to be recognized. It is certain, however, that the two processes are not essentially different, but that the one is the cause and the successor of the other, and that, in the great majority of cases, both are present at the same time. Only early and quite acute cases of miliary tuberculosis may give us the picture of pure tubercle. Caseous bronchitis is essentially a chronic disease, but on careful examination true tubercular nodules will always be found. The nodules lie in the follicular tissue already referred to, between the epithelium and the circular layer of muscle-fibers. These nodules are not infrequently arranged in groups. Rindfleisch * gives a description of such a group of nodules, which has just broken through the epithelium and caused the first loss of substance. The nodules are destitute of blood-vessels, tend to undergo caseation, and on rupturing discharge their contents on the surface of the mucous membrane. In this way the tuberculous ulcer originates. On account of its resemblance to a lentil, it is often called the "lenticular ulcer." In the beginning of its development, it is, however, much smaller than a lentil. This ulcer shows very little tendency to heal and a much greater tendency to spread downward and laterally. There is also a special tendency to follow the course of the lymph-vessels in this extension. In and around the lymph-vessels the tuberculous infection goes deeper, and thus, passing through the thickness of the mucosa, reaches the peribronchial tissue. Peribronchitis is thus produced, in which we find a hard tissue, very rich in cells, cell rudiments, and cell nuclei, in which scattered groups and masses of tubercles are found.

Some pathologic findings clearly indicate that this tuberculous process may develop from the mucous glands. The gland in such cases is infiltrated for the greater part, while some acini are still preserved and the neighboring follicular tissue appears intact. These tubercular nodules which are formed in the mucous glands are characterized by the fact that they are more deeply situated than those that originate in follicular tissue. They produce a widening of the excretory ducts, and at the orifices the ring-shaped erosions, which have long been known to pathologists, and are not easily confounded with the lenticular ulcers due to the rupture of tubercular nodules.

In this connection it may be mentioned that **leprosy** also occurs in the bronchi, and presents similar manifestations—namely, nodules and infiltrations, both tending to the formation of decomposition products and to ulcerations of the free surface of the mucous membrane. The clinical picture is that of a slowly progressing phthisis.

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* v. Ziemssen's "Handbuch," vol. v, 2d ed., p. 192.

SYPHILIS OF THE BRONCHI.

LITERATURE.

"Virchow's Archiv," vol. xv, p. 310.

Wagner: "Archiv der Heilkunde," iv, p. 222, 1863.
Case of diffuse syphiloma of the larynx, trachea, and bronchi.

Verneuil: "Syphilitic Stenosis of the Trachea, and of the Left Bronchi," "Union Med.," No. 29, p. 462, 1866.

Gerhardt: "Deut. Archiv für klin. Med.," vol. ii, p. 541, 1867.
Ulcer in the right bronchus, numerous dilatations, death from erosion of a branch of the pulmonary artery.

Kelly: "Trans. Path. Soc.," vol. xxiii, 1872.
A parallel case: Syphilitic ulcer of the right bronchus at the first point of division; erosion of pulmonary artery; death.

Pye-Smith: "Syphilitic Interstitial Pneumonia." Exactly at the bifurcation the two bronchi were constricted and distorted. The right lung is practically intact, although presenting a general emphysema. The left is densely consolidated, and presents interstitial proliferations and bronchiectases. The bronchial and mediastinal glands are not involved. "Trans. Path. Soc. of London," vol. xxviii, p. 334, 1877.

Schnitzler: "Pulmonary Syphilis and Its Relation to Pulmonary Phthisis," Vienna, 1880.

Ewart: Interstitial peribronchitis, extending along the larynx into the bronchi, especially into the left, which is reduced to the size of a goose-quill. No dilatations. "Trans. Path. Soc.," 1880.

Kopp: "Syphilis of the Trachea and of the Bronchi," "Deut. Arch. für klin. Med.," 1883, vol. xxxii, p. 305.
Two cases in which, in addition to the bronchi, the trachea and lung were markedly involved.

Silcock: "Trans. Path. Soc.," vol. xxxvii, p. 115, 1886.
Syphilitic ulcerative bronchitis.

Packer: Ibid., p. 119.
Enormous constriction of the main bronchi by a thickening of the mucosa. The constriction extends some distance into the smaller branches. In the lungs distinct interstitial proliferations, and in the peripheral bronchi and in the alveoli bronchiectases were found.

Schech: "Contributions to the Study of Syphilis of the Larynx, Trachea, and Bronchi," "Intern. klin. Rundschau," 1887.

Gullion: "Trans. Path. Soc.," vol. xl, 1889.
Described the syphilitic ulcer at the lower end of the trachea and initial portion of the bronchi, causing a narrowing, especially of the left bronchus.

Lanceraux: "Syphilis of the Air-passages," "Sem. méd.," 1891, No. 1.

Favraud: "Syphilitic Stenosis of the Bronchi," "Jour. de méd. de Bordeaux," May 31, 1891.

Kernig: "Petersburger med. Wochenschr.," vol. xviii, 1891.
Syphilitic stenosis of a right bronchus. Respiratory movement abolished. Falsetto tone during phonation (Punchinello's voice). Cough is accompanied by hissing sound.

The text-books on Syphilis.

The occurrence of **secondary syphilis** in the bronchi is not firmly established. Since diagnosis by inspection is for obvious reasons impossible, we can only state that Schnitzler and others have seen in secondary syphilis of the trachea slightly elevated, red patches, which rapidly disappear, and thus resemble a macular or papular eruption on the skin. It is to be assumed that similar changes appear on the mucous membrane of the bronchi. They cannot produce very marked symptoms, for although text-books on syphilis mention that in the secondary stage of that disease the patients occasionally complain of a feeling of tickling in the throat and have some cough and expectoration. Gintrac speaks of a syphilitic cough.

In the trachea papules have been seen by Seidel and Mackenzie. Schnitzler observed catarrh which developed with roseola and abated under antisyphilitic treatment, as well as catarrh which was evidently the continuation of a specific laryngitis and tracheitis. We cannot, therefore, doubt that there is a secondary syphilitic bronchitis. It disappears at the same time with the other symptoms of the disease and, so far as we know, has no practical importance.

Tertiary syphilis of the bronchi, like all other tertiary manifestations, may appear very early or very late; nothing is known of any characteristic beginning. It is associated most frequently with syphilis of the pharynx and larynx, then with that of the lungs, testicles, liver, and spleen (Marfan). The lesions are located most frequently in the neighborhood of the bifurcation of the trachea, or they may be directly continued from the lower end of the trachea, which is a relatively frequent localization of syphilis.

An infiltration soon develops about the gumma, which is circumscribed and ranges in size from that of a lentil to that of a walnut; it may be single or multiple, or diffused over a wide area of the mucous membrane. It does not remain limited to the mucous membrane, but attacks the entire wall of the bronchus. Even the cartilage may undergo a peculiar sclerosis. The infiltrated tissues break down, the extent and depth of the degenerative process being as variable as the infiltration itself. When the degeneration extends to a great depth, healing is attended with the formation of disfiguring scars. Accordingly, syphilitic stenosis, which is such a dreaded result, may develop in two ways: First, by syphilitic infiltration. The mucous membrane is enormously thickened, even to eight or ten times its normal thickness, the epithelium is destroyed, and the underlying tissue is converted into a fibrous, thickened, and sclerotic connective tissue, presenting here and there accumulations of round cells which are not very sharply delimited, gradually fading away into the surrounding tissue. The mucous glands are also destroyed by the growth, which even attacks the cartilage and the tissues beyond it, with the production of syphilitic peribronchitis, and sends out numerous processes into the interstitial tissue of the lungs. This process gradually merges into the second stage. The infiltrate loses its cellular elements, part of it undergoes cicatricial contraction, and the rest is destroyed by ulceration. But the danger of stenosis is only increased, for now a scar-like connective tissue is formed, which constricts and distorts the bronchi more firmly than before. The constrictions may be of the most varied form and extent.

Lanceraux describes also a primary chondritis and perichondritis, without disease of the mucosa or submucosa, in consequence of which the walls collapse and form an extremely hard and narrow tube, which may attain a length of 1 cm.

As the disease frequently attacks the peribronchial tissue, a syphilitic peribronchitis and interstitial pneumonia of greater or less extent almost always develop as complications of syphilis of the bronchi.

Two varieties have been distinguished: *Circumscribed* infiltration of the gumma, which leads to the formation of ulcers and scars; and *diffuse* infiltration, which leads to atrophic shrinking of the mucosa and diffuse peribronchitis. To separate these two forms clinically may not now be possible, as they often merge into one another. But there must undoubtedly be a difference in the symptoms and clinical course, when one or the other form especially predominates. The first form causes more hemoptysis and is limited to the large bronchi; the second causes no hemoptysis, follows the course of the bronchi in its growth and invades the lungs, and leads to considerable cirrhotic change.

Symptoms and Course.—The disease begins very insidiously; there is some cough, but the general impression is merely that of an ordinary bronchitis. At times, however, the attention of the physician is early directed to a surprising admixture of blood in the sputum, which suggests the thought of phthisis. Gradually the symptoms of stenosis are added, and these are often remarkably relieved when expectoration becomes more abundant and more bloody, while they become worse when it is scanty and exclusively mucopurulent. In other cases the symptoms of stenosis do not develop, a hectic fever appears, and the disease assumes the character of phthisis. Hence the term "phthisis syphilitica" has been rightly used. Not rarely it is complicated with tuberculosis, and the basal syphilitic character of the affection is wholly lost. In a certain number of cases other complications give to the disease a peculiar stamp; these are bronchiectasis, gangrene of the lungs, and bronchopneumonia; edema of the lungs is even to be feared.

Diagnosis.—Early diagnosis of the disease is not difficult if the history is carefully inquired into, and syphilis of the pharynx or trachea is evident. But when the latter are free from disease, the diagnosis becomes difficult, although it may now be established much more easily than in former times, as tuberculosis can be excluded on the ground of negative results in our search for bacilli. Where the disease is complicated with tuberculosis, the part of the pathologic picture contributed by syphilis is very difficult to establish. These cases have not hitherto been sufficiently studied by clinicians, and we recommend them to their attention.

All diseases that cause symptoms of stenosis, whether unilateral or bilateral, may lead to confusion. The latter range themselves under the symptom-complex of tracheal stenosis. We may especially mention aneurysm of the aorta and of the large arteries, and tumors of the mediastinum.

The **treatment** consists in the use of mercury and potassium iodid combined. The danger of this affection is so great that treatment must never be delayed or abandoned for some other consideration. Even a perfectly developed stenosis is no counterindication, for in the neighborhood of the old scar there are usually more recent cicatricial processes that are capable of retrogressive metamorphosis. The stenosis may not be improved by tracheotomy; but from the openings made attempts have been made to dilate the constriction through the tracheal wound, and these attempts should be recommended in appropriate cases.

TUMORS OF THE BRONCHI.

Tumors of the bronchi are extremely rare occurrences. The following instances are found in the literature:

1. A case of submucous lipoma, which almost completely filled the left main bronchus. Rokitansky: "Lehrbuch der pathologischen Anatomie," vol. III, p. 25, 1861.
2. A case of colloid struma in the right main bronchus. A tumor the size of a hazelnut, which is regarded as arising from an aberrant portion of the thyroid. The consequence was dilatation of the bronchial branches, cavity-formation, empyema. Radestock: Ziegler and Nauwerck, "Beiträge," vol. III, 1888.
3. An intrabronchial chondroma in a woman sixty-one years of age, occupying the lower branches of the bronchus that supplies the middle lobe. The latter was narrowed and behind the constriction was found a large cavity, the dilated bronchus, into which the tumor projected. It was a chondroma, growing from the bronchial wall, which had undergone partial calcification; in the connective-tissue part the system of blood-vessels was considerably widened, hence a chondro-angioma. Siegert: "Virchow's Archiv," vol. CXXIX, p. 419.
4. Papilloma at the bifurcation of the trachea. Siegert: "Virchow's Archiv," vol. CXXIX, p. 413. Accidentally discovered at the autopsy; the tumor projected into the right bronchus and apparently filled the trachea, but there was no history of dyspnea during life.
5. Chiari*: A mixed lipomatous, chondromatous, and adenomatous tumor which had encroached on a bronchiectasis.
6. The same*: A tumor consisting of glandular tissue, with cavities containing mucus, also encroaching on a bronchiectasis.
7. Macintyre† operated on a patient for papillomatous growths in the larynx. After these were removed, and laryngoscopy revealed nothing abnormal, asthmatic attacks still continued, at the height of which a large amount of blood and a fragment of papillomatous tissue were expectorated.
8. Laboulbène, cited by Chiari: A lipoma which contained spindle-shaped cells (unstriped muscle-fibers?).
9. Williams, in "Trans. Path. Soc.," vol. XXIV, observed a cancerous tumor in the mediastinum, in which peculiar villous outgrowths of the bronchial mucosa, chiefly from the right lung, had formed, causing dilatation of the bronchi. The right bronchus was so narrowed that the respiratory murmur in the lower part on the right side was entirely lacking.

Only of carcinoma have I been able to collect a number of cases:

Ebstein: "Deutsche med. Wochenschr.," 1890, No. 42. Two cases.
Handford: "Trans. Path. Soc.," London, 1889, vol. XL. Primary carcinoma of the left bronchus.

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* *Prager med. Wochenschr.*, 1883, No. 51.

† *Journal of Laryngol.*, vol. VII, from *Centralblatt für Chirurgie*, 1893, No. 50.

Dorsch: Dissertation, Tübingen, 1886. "Ziegler's Beiträge," vol. II. Primary carcinoma of the right bronchus.

Körner: "Münchener med. Wochenschr.," 1888, No. 11. Carcinoma of the right bronchus. Atelectasis of the lung from the obstruction.

Rokitansky says in the "Lehrbuch," vol. III, p. 26: "In the bronchi we observe at times a cancerous process beginning in the bronchial trunk and extending to its branches; the walls of the bronchi are thickened and rigid, the lumen diminished, and the inner surface of the bronchial walls appears rough and uneven. The process occurs in association with carcinoma of the bronchial glands and extensive carcinoma of the costal pleura, to which it is no doubt secondary." At the present time the process would unquestionably be interpreted as exactly the reverse of this.

Reinhard: "Archiv für Heilkunde," 1878, vol. XIX, p. 369, describes a carcinoma which, Birch-Hirschfeld declares, probably developed from the epithelium of the bronchial mucous membrane.

Stilling: "Virchow's Archiv," 1881, vol. LXXXIII, p. 77, reports five cases of cancer of the lungs, at least one of which, the first, certainly developed from the large bronchi. He inclines to the opinion of Birch-Hirschfeld that all pulmonary cancers develop primarily from the bronchi. He describes in detail how the cancerous growth advances, on the one hand, into the bronchi; and, on the other, into the lymph-vessels of the bronchial wall.

Ehrich: Dissertation, Marburg, 1891, describes three cases of pulmonary carcinoma, in one of which at least, the second, the point of origin was probably the right main bronchus.

Langhans: Primary carcinoma of the trachea and bronchi. "Virchow's Archiv," vol. LIII, p. 407.

Chiari, *l. c.*: In the hilus portion of the left lower lobe of the lungs a tumor was found which extended into the lungs along the course of the bronchi. It consisted of papillary growths, covered with tall cylindric epithelial cells, which appeared to have developed from the bronchial epithelium. Metastases were found in the pleuræ, in the bronchial glands, in the cerebrum, and in the liver and spleen.

Bennet: "Trans. Path. Soc.," vol. XXII. Carcinoma of the mammary gland, with metastases in the lungs, exactly following the course of the bronchi. The bronchi were widened, at times markedly sacculated, and the walls were thickened; the bronchial glands free.

Bristowe, "Trans. Path. Soc.," vol. XIX, p. 228, describes a case of carcinoma of the cardia and lower part of the esophagus. Here there was a metastases into the root of the lung, from which the tumor mass followed the ramifications of the bronchi and blood-vessels, so that the finest branches were embedded in it. In the mucous membrane of the bronchi there were small opaque areas. The bronchial glands were converted into tumors.

Rebitzer: Münchener Dissertation, 1889. Case 22. Carcinoma of the central portion of the esophagus. The trachea from the bifurcation upward was filled with yellowish-white carcinomatous nodules, or degenerated carcinomatous masses. The left main bronchus was in a similar condition. Free particles of the new growth were found in both main bronchi.

The last cases, evidently secondary, have no importance for our purpose. In the first group, also, the origin is not always quite clear. But we have a certain number of cases of indubitable bronchial carcinoma, and they will quickly increase if further attention is given to the subject. The view has even been advanced that the majority of pulmonary carcinomata are to be regarded as arising from the bronchi. We will not here follow this theoretic contention, but in practice we can distinguish tumors which arise from the large bronchi and present bronchial symptoms from those which are located in the lungs and in which pulmonary symptoms (lobular infiltration, exudate in the pleura) dominate the pathologic picture. With regard to these conditions, Ebstein also distinguishes the form which remains chiefly limited to the bronchi from that which spreads more extensively to the lungs. The latter will not be considered here. The former are always characterized by the fact that occlusion or obstruction of the large bronchi is present. Aside from the characteristic symptoms of this condition (compare Bronchostenosis), the following **symptoms** are also observed: The sputum is generally catarrhal, but now and then it contains a small amount of blood. Swelling of the lymph-glands occurs in the neck as well as in the axillary space, and is, of course, a very important sign. The cachexia and occasional slight febrile disturbances are also to be considered. The pain is mostly of a peculiar obstinacy and violence, and easily takes on the character of well-defined neuralgia—intercostal, phrenic, or neuralgia of the aortic or of the pulmonary plexus. The disease either remains entirely latent, or it presents the symptoms of pleurisy or of extensive infiltration of the lungs.

BRONCHIECTASIS.

For the older literature, see Riegel or Biermer, "Virchow's Archiv," vol. xix.

Thissen: "Beobachtungen über Bronchiektasie," Dissertation, Würzburg, 1879. Forty-two cases (32 in males and 10 in females). The left lower lobe is most frequently attacked, and then the right.

H. Müller: "Zur Entstehungsgeschichte der Bronchialerweiterungen," Dissertation, Halle, 1882. At the lower end of the left main bronchus is a tumor the size of a cherry, which imperfectly closes both branches. Considerable areas of bronchiectasis.

Richardière: "Centralblatt für klin. Medicin," 1885, p. 335. A three-year-old girl with pleuritis, atrophy of the lung, and extensive bronchitis.

Adelmann: "Petersburger med. Wochenschr.," vol. xvi, p. 102. Bronchiectasis after whooping-cough.

Ronde: "Ueber die Aetiologie der Bronchiektase," Dissertation, Würzburg, 1886. A case which speaks for the etiologic significance of pleuritis.

Bamberger: "Veränderungen der Röhrenknochen bei Bronchiektase," "Wiener klin. Wochenschr.," 1889, No. 11.

Nicaise: "Dilatation des bronches," "Revue de Méd.," 1893, No. 9.

Cayley: "Trans. Path. Soc.," vol. xxxv, 1884, p. 12. The lower lobe of the left lung was the seat of considerable bronchiectasis. In the brain two abscesses were found: one in the right centrum semiovale, reaching to the upper parietal convolution, the other in the left temporal lobe.

William Ewart: "Treatment of Bronchiectasis by Posture and Exercise," "Lancet," July 18, 1901.

A. A. Kanthack and H. D. Rolleston: "Bronchiectasis in Children," "Trans. Path. Soc.," 1897.

Dr. Howard Tooth: "Multiple Cavities in Broncho-Pneumonia," "Trans. Path. Soc.," 1897.

Dr. Walter Carr: "Bronchiectasis in Children," "Practitioner," Feb., 1891.

Dr. Sharkey: "Acute Bronchiectasis," "St. Thomas' Hospital Reports," vol. xxii.

Gerhardt: "Die Rheumatoiderkrankungen der Bronchiektatiker," "Deut. Archiv für klin. Med.," p. 1, Bd. xv, 1875.

Finlay: "Clinical Remarks on a Case of Bronchiectasis treated by Incision and Drainage," "Brit. Med. Jour.," vol. ii, p. 807, 1888.

Byrom Bramwell: "Clinical Studies," June, 1889.

Fagge and Pye-Smith: "Principles and Practice of Medicine," 3d edit., 1891.

Seymour J. Sharkey: "Acute Bronchiectasis," "St. Thomas' Hospital Reports," vol. xxii, 1892.

Sir T. Grainger Stewart: "On the Treatment of Bronchiectasis," "Brit. Med. Jour.," June 3, 1893.
William Ewart: "Two Lectures on Bronchiectasis," "Clinical Jour.," Feb., 1894.
Colin Campbell: "The Treatment of Respiratory Affections by Means of Large Medicinal Injections through the Larynx," "R. Med.-Chir. Soc. Trans.," vol. LXXVIII, p. 93, 1894 *et seq.*, 1895.
Francke: "Bronchiectasis," "Deutsches Archiv für klin. Med.," Bd. LII, 1894.
Laycock: "Bronchiectasis," "Edinb. Med. Jour.," March, 1865.
Sir T. Grainger Stewart: "Dilatation of the Bronchi or Bronchiectasis," "Edin. Med. Jour.," July, 1867.
Sir T. Grainger Stewart and Dr. Gibson: "Twentieth Century Practice of Medicine," vol. VI, 1896.
C. Brian Dobell: "A Case of Bronchiectasis treated by Inhalation of Coal-Tar Creasote Vapour," "Brit. Med. Jour.," June 20, p. 1502, 1896.
Compare the literature on syphilis, on diseases of the bronchial glands, and on foreign bodies.

Dilatation of the bronchi is not a very rare condition, when we consider all the lighter grades of it which occur in the most various diseases of the lungs. A certain degree of localized bulging or dilatation of the bronchi is observed in every condition associated with marked alteration of the pulmonary tissue. As long as this is of slight extent, it has no clinical significance, but as no sharp line can be drawn, these insignificant forms demand some consideration; they are necessary to a comprehension of the condition. In describing the symptoms, the clinical picture will be sketched, but it is by no means explained by the pathologic picture.

Varieties.—There are so many different forms of bronchiectasis that it is impossible to describe them under one heading. I believe that the great confusion which now prevails is due to the grouping together of different forms, and that, of all things, we must have a good classification in order to gain a clearer understanding of the subject. But there lies the difficulty. In this treatise I shall make the following subdivisions:

1. *Inflammatory bronchiectasis*, usually called simply the saccular forms. As the shape is not a determining factor in this classification, I prefer not to use names based on the shape of the dilatation in the clinical description.

2. *Vicarious bronchiectasis*, usually called cylindric, because the dilatation of the bronchi is mostly cylindric in shape; the inflammatory changes in the lungs, when present, do not affect the bronchi.

3. *Congenital bronchiectasis.*

4. *Atelectatic bronchiectasis.*

There is in the literature a large number of bronchiectatic cases which we shall not be able to classify. Not infrequently we find them in the histories of patients, who do not make the bronchiectasis the subject of their complaints, and in whom the expression of the bronchiectasis is transient, so that we can get no idea at all of the character or genesis of the condition.

The division of bronchiectasis into hypertrophic and atrophic forms was tried by the first writers upon this condition. Biermer distinguishes bronchiectasis with inflammatory thickening of the walls, bronchiectasis with simple atrophy of the walls, and mixed forms.

The cases of simple atrophy must, in my judgment, be set apart, for they are found most perfectly when no inflammation is demonstrable or even probable. I have, therefore, formed my second subdivision of these cases. The cases usually seen in practice and diagnosed as bronchiectasis belong to the first or third group of Biermer's classification. The inflammation in these cases is quite regularly present, and its products are distinctly demonstrable in various portions of the bronchial wall and the surrounding pulmonary tissue.

The distribution of the bronchiectasis in the different parts of the lungs has been studied by Lebert, and I quote his figures and add the cases of Trojanowski:

Right lung	15–40	Left lung	13–42
Upper lobe	4–14	Upper lobe	2–10
Middle lobe	1– 2	Lower lobe	2–14
Lower lobe	6–11	Entire lung	9–18
Both lungs	26–35		
Middle and lower lobes	1– 3	Symmetric	15–14
Entire lung	3–10	Predominating in the right	3–10
		Predominating in the left	8–11

But these figures are worthless, as very different processes have no doubt been grouped together.

INFLAMMATORY BRONCHIECTASIS.

Pathologic Anatomy.—The older pathologic anatomy, which was founded by the French writers, subdivides bronchiectasis, in the first place, according to its shape. But this mode of differentiation is decidedly no longer satisfactory; unfortunately, we have not yet agreed on any other. Inflammatory bronchiectasis may assume any shape; the *saccular* is the most frequent, but the *fusiform* and the *cylindric* may also be inflammatory. The latter practically represent the rule in the other forms of bronchiectasis, and so it happens that in practice we occasionally use the term saccular for the inflammatory, and cylindric for the non-inflammatory form. However, I shall not adopt that nomenclature.

In the cylindric form the affected bronchial branches retain the size they had at the beginning, without becoming constricted in their course; they may even increase in diameter. Often the dilated portion passes quite suddenly, rarely gradually, into a tube of normal caliber. Just as suddenly, a tube of much larger diameter may originate from one of smaller size. This form is found mostly in tubes of the third and fourth order. But if the terminal bronchi are attacked, which may occur after infarcts and bronchopneumonia, the section shows the appearance of cavernous tissue. The fusiform is only an accessory form of the cylindric; when several spindles lie together along a bronchus, we speak of *moniliform* bronchiectasis. This occurs especially in the apices of the lungs. Cruveilhier has distinguished enlargement of the whole circumference (*dilatation circonférentielle*) and lateral ectasis (*dilatation ampullaire latérale*), but the distinction has no special value.

Clinically, the most important form is the saccular; this also belongs rather to the smaller than to the larger bronchi. The position of the sacculation is usually such that the air can enter from the trachea, but cannot escape. The passage then may lead through normal, through enlarged, or through narrowed bronchi.

When the terminal portions of the bronchi, the infundibula, become enlarged, the alveoli opening into them participate in the process, their septa atrophy, and small cavities are produced which have been designated pulmonary vacuoles. They may be quite similar to the dilatations occurring in emphysema. In many cases the sac is not only without exit, but the bronchus leading into it is also obliterated, so that the result is the formation of a cyst. The size and number of the sacs may be quite different. Cavities have been found of the size of a hen's egg, although they mostly vary between the size of a cherry and that of a walnut; the smaller are, as a rule, more numerous and located at the periphery, while the larger are isolated and centrally placed. We have cases where the whole lung is changed into a spongy tissue. [In the saccular form the pulmonary tissue of the whole lobe of the lung may have disappeared for areas of considerable extent, and in its place large numbers of rounded, smooth-celled, shiny saccules are seen, each presenting a small opening in its floor. This constitutes the condition known as the "*turtle lung*," from its similarity in appearance to the lung of that animal.—Ed.] We often find cylindric, fusiform, and saccular cavities together, and can see how the one form may pass over into the other.

The bronchial wall in the ectatic portions is never normal. In the mildest cases the wall is in a catarrhal state, the epithelium is preserved only in patches, the mucous membrane is thickened by cellular infiltration, and the normal elasticity is diminished or wholly lost through its influence. If the catarrh persists for some time, it leads to hypertrophy. The mucous membrane is so uneven that it appears to be covered by small villi, and the glands and cartilage lying under it are likewise hypertrophied; in this hypertrophy even the connective tissue of the external bronchial wall takes part, and it grows from there into the alveolar septa. After a certain time the connective tissue undergoes a cicatricial contraction; the swelling of the mucous membrane gives way to atrophy, the membrane is no longer smooth, but very uneven; and wheals are produced at various points on the bronchial wall. Sometimes the cartilage remains enlarged, although in other cases it certainly atrophies with the other tissues. The muscular and elastic tissue in part become irregularly atrophied, and the connective tissue projects into the lumen and forms trabeculæ. Accordingly, the condition is also called trabecular degeneration of the walls.

The connective tissue in many cases takes the place of the longitudinal elastic fibers, and a longitudinal striation thus arises; if it replaces more the transverse fibers, a transverse striation develops; finally, if it develops in both directions, it gives rise to a reticular appearance. Hypertrophied muscle-fibers have also been found in many cases. The result is always that the bronchiectatic areas have a thick wall. But there are also cases in which the enlargements have quite a thin wall, where the change is accompanied by the process which we know as atrophic catarrh. This form may occur as the terminal stage of the process just described, but in that case only a portion of the bronchial wall atrophies; there are always thickened masses and interstitial processes, which extend through the lungs in strong bands of scar tissue and constitute a characteristic feature of this variety of bronchiectasis. The epithelium is frequently said to be well preserved, because ciliated epithelium is often obtained in bronchiectasis. There is no doubt, however, that its continuity is destroyed to a considerable extent, or else the type is changed to the pavement epithelium which we know in bronchitis. Nevertheless it is quite true that, on careful search, in protected places, we may often find very well-preserved ciliated cells.

The walls of the bronchi often undergo more extensive and severer changes. In the first place calcifications may form, which preferably attack the cartilage, but are also found in the connective and muscular tissues; particles of lime are even found in the epithelium. In the second place, superficial ulcerations form, which may become covered with a peculiar grayish-yellow exudate of offensive odor. These ulcerations may then give rise to cicatrices, to connective-tissue growths, and to shrinking of the tissues. But they may also be the point of origin of pulmonary gangrene. In the third place there may be a considerable change in the blood-vessels *; these may become symmetrically enlarged, so that a cavernous meshwork lies in the submucosa, or they may form numerous small aneurysms.

Adhesive processes and deformities of the bronchial wall often occur. We have already mentioned that the bronchi leading from the dilatation are quite usually obliterated. The same may happen to the bronchi leading to the enlargement, and thus the bronchiectatic cyst is formed. The contents may consist of a purely serous fluid, a more exact analysis of which has not yet been made. The contents may become inspissated or calcified even to the formation of concretions.

[According to Ewart, the distinction between tuberculous cavity and simple dilatation occurring at the apex never presents any difficulty, except in chronic cases of phthisis where a vomica has emptied all its matter and presents a smooth and relatively dry surface. Close inspection will show: (1) That the bronchus opens into the cavity too abruptly for bronchiectasis; (2) that the bronchial membrane can only be followed for a short distance around the orifice of the bronchus; and (3) that the wall of the cavity presents none of the

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* Hanot et Gilbert, *Arch. de physiol.*, 1881.

sculptural detail which identifies the original structure of a bronchus even in extreme dilatation.—Ed.]

The appearance which a bronchiectatic lung presents is very variable, according to whether the dilatations are large and single, or small and multiple. In the latter case the lung may be likened to a sponge filled with purulent mucus. A single dilatation is generally larger and situated near or in the interior of the lung, while multiple ones are more superficial; but there is no hard and fast rule. [Speaking generally, it may be stated that when the condition follows acute or chronic bronchitis, or bronchopneumonia, especially in childhood, many tubes are dilated; whereas when it is a sequel of collapse from pleural effusion, lobar pneumonia, the impaction of a foreign body, or stenosis of a large bronchus, it may for a time be limited to the area of lung affected by the primary lesion.—Ed.]

The secretion in the dilated bronchi and in the sacculations consists of mucus, containing large numbers of pus-cells and desquamated cells from the epithelium of the bronchi and alveoli. The pus may through infection become putrid; in places where the communication with the air is limited, it may become inspissated, fat needles may crystallize out, calcium salts may precipitate, and friable, mortar-like masses may be formed, or the process may even go on to the formation of broncholiths. Blood is also occasionally found; and, of course, different kinds of bacteria and fungi. Under other circumstances fat crystals may separate as in putrid bronchitis, or cholesterin may be formed. [In some cases the dilated bronchus throughout its length is filled with clear, sweet, gelatinous-looking mucus, somewhat resembling the contents of a culture-tube of nutrient gelatin. On closer inspection it will be seen that the upper surface presents a yellowish tinge, and here a growth of micro-organisms may be found, rendering the likeness still more complete.—Ed.]

When the dilatations are shut off from communication with the air by the obliteration of the proximal portion of the tube through which the air should enter, they often contain a light-colored, clear mucus. The ordinary purulent forms act like true abscess cavities. We find in them no epithelial covering, that might suggest bronchial epithelium, but a layer of fusiform cells, with scattered pus-corpuscles. The elastic tissue lying under the epithelium is often no longer to be found, or is only rudimentary, so far as it is present; the muscle-cells may also have disappeared, and in some places be replaced by connective tissue; in other places, however, they are found, and even appear hypertrophied. The cartilage also has suffered from injurious influences, becomes calcified and sloughs out, or becomes atrophied. It is very surprising how differently the various portions of the wall of the cavity may act; evidently the atrophic and hypertrophic processes change according to the influences of pressure, degeneration, and inflammation. The same may be said of the glands. Cornil and Ranvier are of the opinion that the region which they call the "equator of the ampulla" shows the most extensive atrophic changes; here the muscle-fibers have entirely disappeared, while they are still preserved at the poles. But this law can be considered as correct only in a very general way, and it has numerous exceptions. The surface of the cavity often shows numerous granulations, inflammatory growths, which may be extraordinarily rich in blood-vessels and in which the vessels present occasional aneurysmal changes. These granulations may be the source of very obstinate hemorrhages.

The lung tissue around the bronchiectatic area naturally shows marked changes: emphysematous conditions and fibrous thickenings are to be observed. The latter have been characterized as cirrhotic, and others, again, speak of sclerotic conditions of the lungs; indeed, this tissue may assume the hardness and toughness of very dense scar tissue. The pleuræ almost invariably participate in the process, when the foci are at all extensive and not entirely limited to the central portion of the lungs. The two layers may become adherent, and a large mass of scar tissue may bind the lungs to the thoracic wall. The bronchial glands also usually participate in the changes. The bronchi, which are not enlarged, and the trachea usually present catarrhal changes in bronchiectasis.

In a certain number of cases tuberculosis complicates the clinical picture of the bronchiectasis; while the one is not excluded by the presence of the other, they do not coexist very frequently. Smaller bronchiectatic areas, which have no special clinical significance, are not infrequent in tuberculosis. There is a tendency to the formation of larger bronchiectatic areas in tuberculosis only in cases which tend to recover by the formation of connective tissue, and these are always very circumscribed processes, occurring in the apices of the lungs. Emphysema of the lungs of a higher degree is often found in the final stage of bronchiectasis; it begins as an emphysema of the borders of the lungs, representing a kind of compensation, to aid in the filling out of the pulmonary space in the thorax; its occurrence is favored by coughing.

Changes in the pleura are very frequent in this disease, so much so that the pleural affection has even been designated as the cause of the bronchiectasis. The same connective-tissue formation which leads to sclerosis of the lungs causes also fibrous pleurisy, resulting in adhesions. The pleurisy does not, however, directly lead to bronchiectasis. Large exudates compress the lungs and the plastic formations that remain render the compression permanent. Degenerations must influence the lung tissue by the formation of scars, but they have not, as a rule, the power to cause bronchiectasis, unless certain other necessary factors are superadded. We shall return to this subject later.

Atelectatic areas are frequent in bronchiectasis, but are only of very slight extent. Larger atelectatic areas never exist long, as they always lead to shrinking, with marked changes in the bronchi and in the lung tissue; they may then occasionally cause the formation of cysts. Atelectasis as a cause of bronchiectasis is still to be taken as a subject for special consideration.

Changes in the Lungs.—The lung tissue in this form always participates by means of an inflammation (and therefore I call it the inflammatory form) or the pulmonary tissue is the part first affected. Charcot describes a special disease as "*sclérose bronchopulmonaire avec dilatation des bronches*"; it is nothing but Corrigan's cirrhosis, except that Charcot regards the bronchiectasis as the necessary result of a specific form of the chronic inflammation of the lungs. This hypothesis is by no means supported, as we do not see in what respect this affection is distinguished from other chronic forms. Charcot himself admits that there are forms in which the sclerosis of the lungs predominates to such an extent that the bronchiectasis plays no essential part, and so Marfan decides to devote a special chapter to bronchopulmonary sclerosis, in which he adds nothing new to what he says under bronchiectasis, but merely establishes the point of view that there is a quite special disease of the lung tissue, the introductory stage of which he describes as "carnization." This condition of the lung tissue evidently occurs during many inflammatory processes, especially in bronchopneumonia, but the bronchi are said to be enlarged from the first. This peculiar form of inflammation has never been recognized by special pathologists, so far as I can see. It is the expression of our clinical necessity for the understanding of this form of bronchiectasis. Thus, the dilatation of the bronchi is here represented as an affection which from the beginning is a peculiar feature of the clinical course, and which cannot be regarded as secondary to the atrophy of the lungs. This conclusion has the very attractive qualities of simplicity and clearness, but it is by no means demonstrated, and really is not even probable. The clinician might, in the cases in which the etiology is still very obscure, be tempted to accept this conclusion as true, for many doubts may be settled with it, but it is still only a theory. When I consider the cases of bronchiectasis due to the aspiration of foreign bodies, we have before us in these characteristic examples a bronchopulmonary sclerosis corresponding in all respects with the descriptions of Charcot and his followers, although there can be no question of anything specific. These cases, on the other hand, suggest the assumption that when the normal course of an ordinary inflammation of the lungs is disturbed by any injurious agents, when cause for new inflammatory processes is given, then the opportunity for formation of bronchiectatic areas occurs. That the opportunity depends on the location and the extent of the inflammation there can be no doubt. If observers will direct their attention to this point in each individual case, I have no doubt that the guiding thread in this labyrinth of symptoms will eventually be found.

Etiology.—The manner in which bronchiectasis develops has given rise to many explanations and many hypotheses. We find these things minutely cited and critically considered by Biermer,* then

* *Virchow's Archiv*, vol. XIX.

again by Jürgensen,* and now by Marfan.† All authors are agreed on the fact that bronchiectasis can only arise after the capacity for resistance of the bronchial tube has been diminished by some destructive process. General nutritive disturbances, such as anemia, malnutrition, or alcoholism, are not in themselves sufficient to cause the condition; there must be some inflammatory process to destroy the integrity of the bronchial structure. All such inflammations are therefore predisposing causes of bronchiectasis.

The ordinary forms of bronchitis evidently do not fulfil these conditions, for otherwise bronchiectasis would be a very frequent occurrence. Marfan says that the bronchopneumonia of influenza, measles, whooping-cough, and typhoid fever predisposes to the condition, but I cannot agree even with this statement; a special malignancy is necessary in these diseases to bring about such consequences, and it is not always perfectly clear on what this malignancy depends. At all events, there is a general agreement of opinion regarding this main predisposing condition.

There is much less agreement concerning the exciting cause. With many others, I find this cause in the increase of pressure in the affected and already diseased bronchi. It is firmly established that in expiration, when the glottis is closed, the pressure may easily rise to 80 mm. of mercury, while in quiet expiration it does not exceed 2 or 3 mm. In this way such enormous variations are produced that we cannot wonder at their exerting an injurious influence when the normal structure of the bronchi is very much altered. For the healthy bronchus can endure a great increase of pressure, as is shown in the case of singers, whose lungs are strengthened by singing.

Crying, singing, and especially coughing affect a bronchial tree with normal powers of resistance in such a way as to cause a general bronchiectasis, which becomes noticeable only when it is excessive, and immediately becomes neutralized. If, on the other hand, there are one or several weak spots in the bronchial wall, variations of pressure may be quite different in their significance. In order to adhere to facts, and not to wander too far in the realm of hypothesis, we must ask in what conditions bronchiectasis is principally found. When we ask for the causes of bronchiectasis, we find all possible pathologic conditions enumerated. I said at the beginning that there were no marked changes in the tissues of the lungs and pleura, in which these enlargements were not occasionally observed. Biermer mentions the following: (1) Bronchitis, especially capillary bronchitis; (2) lobular catarrhal pneumonia; (3) typhoid hypostatic conditions; (4) lobar pneumonia, especially of subacute or chronic course; (5) tuberculosis; (6) obliteration of the bronchioles, with slaty induration of the surrounding tissue; (7) stenosis of the larger bronchi (by cicatricial contraction, compression from without, or obstruction from within); (8) marasmus of the lungs; (9) pleuritis and pleural growths. It is clear that such an enumeration is of extremely little value for an insight into, and thorough understanding of, the matter.

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*In Ziemssen, "Sammelwerk."

†In the new French "Traité de Médecine."

I find one factor, which may be mentioned here as preeminently frequent: stenosis.

For further illustration, it is well to remember that Lichtheim was able to produce bronchiectasis experimentally.

According to Lichtheim, Traube has proved experimentally that artificial occlusion of a bronchus produces atelectasis of the portion of lung tissue supplied. Lichtheim himself confirmed the experiment by a method which is above criticism *; twenty-four hours after total occlusion of a bronchus, the lung tissue which it supplies becomes absolutely atelectatic. The bronchi in the atelectatic portion on the distal side of the occluding plug were filled with thick, white pus. But when the experiment was performed in such a way that the animal continued to live some weeks after occlusion of the bronchus, there resulted a moderate dilatation of the entire bronchial tree, leaving a portion of the lung tissue atelectatic; or the bronchial system was gorged with tenacious white pus while the pulmonary parenchyma was reduced to a minimum. The latter represented the greatly attenuated, indurated wall, barely a millimeter in thickness, of a pus-sac which on section presented a number of pockets communicating with one another by large openings. The appearance of the specimen strongly suggested that of a kidney with a greatly dilated pelvis as the result of pyelitis. One of Lichtheim's cases possesses special importance. It should be stated that his operation was always followed by empyema. In this particular instance the lung was found atelectatic but otherwise uninjured, surrounded by the pus of the empyema. If this means anything, it means that occlusion of a bronchus does not necessarily cause bronchiectasis in the portion of lung supplied, unless there is at the same time an opportunity for the development of a purulent inflammation in the bronchi, for Lichtheim regularly found pus in the dilated bronchi. If this conclusion is correct, stenosis must favor the development of bronchiectasis even more than absolute occlusion of a bronchus. For if the occlusion is aseptic, it will result merely in atelectasis and atrophy of the corresponding portion of the lung, whereas partial occlusion will permit the entrance of air, and with it, in all probability, the accidental introduction into the bronchi of germs, which, owing to the difficulty or impossibility of their expulsion, become the cause of inflammatory processes.

Now we can see how extremely complicated the question is, and why in different cases, even under apparently very similar conditions, the results may be quite different.

The experiments teach the following facts: In an inflamed and occluded bronchus the secretion collects so as to cause a very considerable increase of pressure, so considerable that it may lead to

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* "Archiv für experimentelle Pathologie und Pharmakologie," vol. x.

bronchiectasis, the dilatation being of course aided by the changes in the bronchial wall resulting from suppuration. But in the majority of cases that come to the autopsy there is no question of such a closure of the bronchi.

In what way, then, does stenosis act, since, according to experience, it so frequently gives rise to bronchiectasis?

During inspiration the air is sucked through the stenosis, but in expiration the expelling force is often insufficient; for the expelling force in this case is represented only by the elasticity of the tissue lying beyond the constriction. The high expiratory pressure, which can be exerted by the action of the expiratory muscles, will in most cases do very little toward expelling the air, as it can never work as uniformly from all sides as the elastic pull, which drives the air into the bronchus during inspiration; it acts intermittently, and must press equally on all parts of the space. It is quite possible that the inspiratory pull sucks air from the larger bronchi through the stenosis into the air that has already been inspired. If a mass of mucus is present at the stenosis, it may work like a valve, so that air can get in much more easily than it can get out; stagnation of the air results, the pressure becomes very high and remains so continuously. Attacks of coughing greatly assist the process; for if the distal bronchus is unfavorably situated, it is much easier to drive air into it from the other portions of the lungs than to force out the air already in it.

In the study of the literature we find that Barth has already said that the shutting off of the inspired air in the bronchi on the distal side of obstructing masses of secretion may be a subsidiary factor. Biermer states that he cannot agree with this; "for, aside from the fact that this isolation of the air has never been observed in bronchiectasis, I do not think it is plausible that the air crowded behind the secretion should be kept imprisoned in dilated bronchi, which are usually filled not with tough but with rather friable secretions." This objection is, however, very weak when we consider that the isolation does not take place in the dilated bronchi, but that the dilatation must be peripheral to the confined body of air; that the nature of the secretion is very variable and that looser secretion arises from the bronchiectatic areas. But how is it with the stenoses on the proximal side of the dilatation? Such isolation of the air has, of course, never been observed, because no attempt has ever been reported and we know of no method for such investigations. I must, then, assert that Biermer has made his rejection much too easily, and that there is much truth in Barth's line of argument.

The portion behind the constriction is thus not only transiently, but in a certain number of cases permanently, brought under the influence of very high pressure, and with it conditions are given which explain why even a thickened bronchial wall may yield and why bronchiectatic areas, which are surrounded by hard scar tissue, become larger and larger. Permanent excess of pressure is a power which, as we see in aneurysms, not even bone itself can withstand.

I therefore consider stenosis and continuous excess of pressure the two factors which work together; stenosis is necessary, because it alone makes intelligible the occurrence of an adequate and continuous excess of pressure. The stenosis will evidently not always be necessary to bring about the conditions. It is only a frequent factor in the process, but it is by all means a very frequent one.

Biermer himself says that stenoses of the large bronchi are rare, but regularly lead to dilatation of the branches lying below the stenosis. Riegel says in regard to the anatomic consequences of stenoses, that the dilatation of the bronchial branches lying below the stenosis is to be especially mentioned. It is very rarely absent in bronchial stenosis, and especially when the stenosis has developed rapidly and is of such high degree that the breathing in the corresponding portion of the lungs is entirely or almost entirely abolished. Marfan says in regard to stenosis of the trachea and of the large bronchi: "There is a dilatation above and below the point of constriction."

Irvine has given an interesting contribution on this question in volumes XXVIII and XXIX of the "Pathological Society Transactions." He describes the changes that took place in lungs after compression of the supplying bronchus by an aneurysm. Such a compression evidently takes place very slowly, so that one cannot make comparisons between these cases and the experimental cases. The isolated lung was enlarged, the pleura was uniformly thickened but the pleural cavity was not totally obliterated, there was no question of a brawny thickening, but the two layers were adherent to a certain extent. The lung consisted of a number of cavities, filled with an odorless fluid containing reddish particles of solid matter, decomposed blood, pus, degenerated lung tissue, and caseous masses. The bronchi had more or less given way; the walls were broken in different places, so that the dilatations formed a portion of the numerous cavities. The author assumes that emphysema is the first consequence of compression. He explains the tendency to excess of pressure and the difficulty of expiration in a way similar to the one given by me. The secretion gradually accumulates, causing an inflammation, and when the bronchus is entirely filled or occluded, the excessive pressure due to the suppuration and consequent destruction of tissue take place.*

Lebert also says that below the stenosis the bronchi are usually dilated, as we have already stated.

But in order not to omit any doubtful statements, I will quote one which is made by the above-mentioned Biermer in another place: "Much more rarely is bronchiectasis associated with stenosis of the larger air-passages"; nevertheless the literature of syphilis contains many cases in which cicatricial stenoses of the trachea were complicated by secondary dilatations of the bronchial branches. We have also observed some cases in which neoplasms, either by exerting pressure from without or by encroaching on the lumen of the bronchus, narrowed the latter and caused dilatation below the constriction. Kopp has two cases of syphilitic bronchial stenosis, in one of which there was a characteristic bronchiectasis, while in the other there was none at all.

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* In the section on Bronchostenosis many cases are given from the literature, which might be cited here.

Cohn, however, says*: In **stenosis** of the bronchi, whether by pressure from without, by constricting scars, or by foreign bodies from within, anatomic experience rarely shows us dilatations behind such constrictions. In experiments which he so conducted that he brought foreign bodies into the bronchi, an ectasis was found around the obstructing body, but not behind it; rather an emphysema developed in the cases of partial occlusion. It is really interesting to find such opposing statements in regard to so fundamental and so simple a question. It is explained by the fact that while it is well known that constrictions have been dissected out behind which there was no enlargement, yet in the overwhelming majority of cases the opposite is the case. I have taken the trouble to look for autopsy cases in which the dilatation behind the constriction was wanting. They are not easy to find. In many cases of constriction it is stated that there was bronchiectasis, and in many no statement is made on the subject. The negative fact that no bronchiectasis was present was not especially mentioned. No particular attention seems to have been paid to this point. But enough facts have been presented to show without a doubt that there are constrictions without secondary bronchiectasis.† The question is why in one case ectasis results and in the other not; this has been considered by Riegel. It all depends on the mode of production and on the effect of the stenosis.

If we try, on the basis of the material collected, to answer the question: How is the effect produced by the constriction of a bronchus? we must first consider especially the effect of sudden occlusion. In respect to that, the experiments are good guides: atelectasis is produced. If the atelectasis was perfectly aseptic, it was followed by shrinking and atrophy; but if there was opportunity for the entrance of the germs of inflammation, suppuration followed, causing increase of pressure with the formation of bronchiectasis and the development of chronic peribronchitis, pneumonia, and pleurisy, and resulting in the formation of scar tissue or even in phthisis, if there was opportunity for infection.

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* "Aus den Abhandlungen der Schlesischen Gesellschaft für vaterländische Cultur," "Abtheilung für Naturwissenschaft und Medicin," 1862, Heft 1.

† Z. B. Kopp, *Archiv für klin. Medicin*, vol. xv; Immermann, *ibid.*, vol. v. Andral's celebrated case of simple inflammatory stenosis is such a one. In that of Ewart, *Pathol. Soc. Trans.*, vol. xxxi, p. 57, the following statement is made: The opening of the left bronchus is reduced to the size of a goose-quill. The left lung was reduced to half its normal size, the tissue tough and uneven. The branches of the bronchi were greatly thickened and surrounded by a zone of thickened and pigmented lung tissue. The lower lobe was swollen and much softened, and could not be entirely separated from the pleural thickenings. Not a word of bronchiectasis.

What occurs more frequently, however, is gradual closure or constriction of the bronchus. It favors stagnation of the air and increase of pressure in the peripheral portion, and so long as the condition remains, at least to some degree, aseptic, the main result will be emphysema. With time, however, germs must get in, their growth is favored more and more; then we find bronchopneumonia and peribronchitis developing. Bronchiectasis develops only: (1) Through stasis of the secretion, when the opening becomes very narrow or is entirely occluded; (2) through stagnation of the air, when the opening remains wide, but may work like a valve, according to the views expressed above.

The fact is, then, that when a stenosis exists we can expect, with a very strong probability of fulfilment, the development of bronchiectasis to follow as a result. But when we have a bronchiectasis, we cannot reason backward and conclude that a stenosis is present. It is certain—and it can readily be seen by a consideration of my collection of the literature on bronchostenosis—that the stenotic form of bronchiectasis is of very frequent occurrence, being present in many cases in which it is not recognized, so that a portion of our cases of inflammatory bronchiectasis might be classed under the stenotic form. The genesis of this stenotic form of bronchiectasis can now no longer lead to any marked differences of opinion. The fundamental cause is the stenosis; the predisposing causes are the pathologic changes which, in connection with the stenosis, develop in the portion of the bronchi lying behind the stenosis, and which are due to inflammation; the determining cause is the increase of pressure in the separated portion, mostly of air pressure, under the influence of respiration,—inspiration as well as expiration,—and especially of the cough, and occasionally of crying and singing. The increase of pressure, for there must be increase of ·pressure, may likewise be caused by stasis of the secretion, and in complete closure of the bronchus there can be only the stasis of the secretion and of the pus, but in partial closure there is often also increase of air pressure.

In this way the form of bronchiectasis which I have called stenotic is explicable. These cases are of very great practical importance, and it seems probable to me that, on further consideration, many cases will be regarded as stenotic which we cannot as yet include in that class.

But it is undeniable that there is a group of bronchiectatic cases where the stenotic explanation would have to be very much forced and where the pathologist at the autopsy finds the ectasis arising directly from normal or even enlarged bronchi. These cases belong, however, to the inflammatory class, because they are found in an inflamed tissue (peribronchitis and pneumonia). Clinically, according to my opinion, this form is not nearly so important as the stenotic; it occurs much more rarely and the symptoms of infiltration and shrinking of the lung tissue are much more prominent than those of bronchiectasis. For their development it is necessary to consider other essential factors of injurious influence. We can select from this group a certain number of cases; we have already distinguished a bronchiectasis from stenosis, and we now separate, as our second subclass of the inflammatory bronchiectases, the induration bronchiectasis, in which a thickened membrane attaches the portion of the lung affected with the ectasis firmly to the thoracic wall.

We see, from the hard masses at the periphery, bands penetrating into the interior of the lungs, and these must also influence the bronchi. It is possible that these bands may lead to a dilatation of the bronchi; but how uniformly it would have to exert its influence on all sides to attain this result! I believe, rather, that a constriction from pressure lies at the bottom of this form also. This pressure acts on the external portion of the bronchus and will usually not lead to a pathologically perceptible stenosis in the cross-section of the lumen, although a closure, or at least a constriction, is really present, somewhat like a kink in a rubber tube. Would not a closer study of this condition rank it among the stenotic cases? The only thing for me to do is to state that hypotheses enough have been advanced, and it is time for the careful investigator to confirm them. In this form of bronchiectasis we have to consider that the lungs remain compressed during the whole attack of pleurisy, and later by the pressure of the exudate, and so their power of respiration is much impaired. In these cases it may very easily happen that air is pressed from the other lung during attacks of coughing on account of compressed places in the bronchi, which are due to the pressure and distortion in the affected lung. This air, which is crowded in under high pressure, cannot get back. Thus opportunity would be given for the development of stenotic bronchiectasis. In certain cases, however, a portion of the region regains the power of respiration, and the necessary pressure in the bronchi may then be attained through aspiration, as already mentioned.

[*Fibrosis of the Lung.*—Fowler and Godlee have come to the same conclusion as Hoffmann with regard to the theory that traction by fibrous bands passing from the pleura to a bronchus, or from one bronchus to another, constitutes the effective dilating force when bronchiectasis follows fibrosis of the lung; *i. e.*, they take issue with those who uphold it. They say that areas of fibrosis, whether of tubercular origin or not, tend to shrink by centripetal contraction. Chronic cavities tend to contract, and their lumen may be almost obliterated. The parietal and visceral layers of the pleura become separated as the lung contracts, and the intervening space is filled with a fluid exudate which ultimately becomes organized into a densely firm fibrous tissue, or the neighboring tissue, if still pervious to air, becomes emphysematous. Organs are displaced and other changes occur, all of which appear more likely to obliterate than to enlarge spaces in the affected area.—Ed.]

Concerning the bronchiectasis from **foreign bodies,** Cohn assumes, as I have already cited, on the ground of experiments, that the dilatation usually occurs around the foreign body and not behind it. In these cases it is the continual irritation that causes the inflammation and ulceration, and thus an ulceration cavity develops. We cannot, however, class this as a bronchiectasis without further investigation. The section of lungs affected by such foreign bodies shows us that not a single large cavity is formed by the foreign body, but that, besides the cavity in which the foreign body lies, numerous others are present. Thus the matter is somewhat complicated. The local irritation acting continuously causes a peribronchitis, which, extending from the position of the foreign body to the bronchi, favors in a special way the formation of bronchiectasis.

For an example, I would call attention especially to the case so exactly described by Kocher.* I must add, also, that foreign bodies are too little considered as causes of bronchiectasis. We have cases enough in which the course and all the circumstances did not suggest the idea that a foreign body could have passed into the lungs, and where the autopsy first showed, to the astonishment of the physician and friends, what was the trouble; or the accidental expectoration of the foreign body established the diagnosis. If the foreign body is small, if it reaches the finer bronchi without special coughing or attacks of suffocation to loosen it, as may easily happen, it may remain there a long time, then gradually crumble and be coughed up with the mucus in microscopic pieces, and so the diagnosis is never made. Smaller pieces of meat or vegetable matter may in this way cause a foreign-body bronchitis, which easily leads to bronchiectasis. If the foreign bodies are so fine that they make their way into the alveoli in the form of dust, then the case is, of course, quite different, and we find that in the real cases of pneunonokoniosis ectasis rarely plays any part. The essential thing in foreign-body bronchiectasis is evidently, again, the condition that the air can during quiet inspiration easily pass the body which has penetrated, whereas in expiration stagnation results which is relieved by an attack of coughing that does not, however, remove the obstruction. This, again, is an example of the stenotic form which has already been mentioned.

Tuberculosis.—Lebert says: "The fact is surprising that in one-sixth of my cases there was spitting of blood, although not in large amounts, yet sufficient so that it could not be confused with the bloody sputum of pneumonia. The first hemoptysis came too early to allow the assumption that it came from hemorrhages in the walls of bronchiectatic cavities."

But how does he explain this tendency of the disease to begin with hemoptysis? Such a beginning is found, as is well known, principally in phthisis. Does a portion of the bronchiectasis develop during beginning tuberculosis, from which the patient later recovers? We can find in all the text-books that the ordinary course of tuberculosis does not lead to bronchiectasis; indeed, in many cases tuberculosis and bronchiectasis are placed in opposition to each other, as if a tendency to the one excluded any tendency to the other. Yet it is, again, established pathologically that a tuberculosis establishing itself in the finer branches of the bronchi may lead to the formation of small bronchiectatic areas, which, however, do not produce clinical symptoms, and are therefore never named in the clinical description of bronchiectasis.* In many cases of very chronic tuberculosis which have been described as fibrous phthisis characteristic bronchiectases, on the other hand, occur. It often remains a question at the autopsy whether the tuberculosis has not established itself in the bronchiectatic spaces, and is thus a complication of the primary bronchiectasis. Yet the course of certain cases of phthisis which have recovered indicates decidedly that the cicatricial contraction of the tissue was really the cause of the bronchial dilatation. This is not at all surprising.

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* *Wiener klin. Wochenschr.*, 1890.

Syphilis easily causes the formation of bronchiectasis, and we can readily understand this, as both scar formation and infiltration tend to produce changes behind which the conditions for the development of stenotic ectasis are especially favorable.

Pneumonia.—Finally, I will mention the chronic pneumonias. Part of these stand in relation with the injuries which I have already mentioned (foreign bodies, syphilis, and pleurisy); another part develops from acute pneumonia, whether croupous or bronchopneumonic in form; a small number is distinguished, finally, as primary, as we do not know their cause. Whether these, however, cause bronchiectasis without one of the predisposing factors mentioned (stenosis, thickened exudate, and foreign bodies) I greatly doubt.

Course.—Bronchiectasis has a characteristic course only in very well-marked cases, and in those in which no special complication influences the progress of the disease. Thus, the occurrence of gangrene or of some other infection must lead to febrile disturbances which are by no means found in the simple form of the disease.

The beginning of the disease is uncertain; an acute or chronic inflammation of the bronchi, lungs, or pleura precedes the onset, and this does not tend to become localized; this, the *first stage*, is apt to be confounded with an ordinary bronchitis; we only notice that the bronchitic symptoms appear especially and persistently in certain circumscribed areas. In the *second stage* the course begins to assume a characteristic type. With a satisfactory general condition, we find a surprising irregularity in the expectoration; it is at times scanty and at times very abundant; there may even be a very characteristic variation, so that at certain times of the day there is an abundant expectoration which is almost or wholly lacking at other times. This condition may last a very variable time and may remain stationary for years. But finally it reaches the *terminal stage*, when the bronchiectatic phthisis develops. The patient becomes emaciated; he is cachectic or cyanotic; fever and night-sweats, loss of appetite, and diarrhea develop, and lead to death. Among 52 cases Lebert and Barth had 13 which lasted for more than ten years; 11 lasted less than a year; 4, one to two years; 16, three to five years; 8, six to ten years. Lebert justly remarks that the statistics are very misleading, and that in the better private practice the cases with very long course, and stationary cases predominate.

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* Grancher shows that they can be considered as the beginnings of cavity formations. *Gazette méd. de Paris*, 1878.

Biermer distinguishes the following clinical varieties as regards the course of the disease: (1) There is the picture of an ordinary chronic bronchitis, but the nature of the expectoration and the physical signs show that there is ectasis. This is the classic form. (2) The picture of fetid bronchitis. (3) Emphysematous habitus, much dyspnea, violent attacks of coughing, venous cachexia, and cyanotic-hydropic symptoms; the autopsy shows bronchiectasis and emphysema. (4) Phthisical pictures, when either the ectasis is complicated by tuberculosis, or the lung tissue is free from tubercles, but shrunken by connective-tissue and pigmentary induration. (5) Combination of pleuritic exudates with bronchiectasis. (6) Combination of bronchiectasis with simultaneous stenosis of the larger air-passages—a very rare case. Suffocative attacks and difficult expectoration play the chief rôle; the bronchiectatic symptoms may be masked.

[The course of a typical case of bronchiectasis, according to Fowler and Godlee, presents six more or less well-defined stages: (1) Expectoration is profuse and purulent, but not fetid; pyrexia is absent. (2) The expectoration acquires a faintly fetid odor and becomes more profuse; this stage may last for some time. There may be slight pyrexia, but it is rarely continuous, being, as a rule, limited to periods when the sputa are diminished in amount or entirely absent. (3) Sputa are decidedly fetid, the physical signs are characteristic, and clubbing of the fingers is well marked. This is the stage of complete development. (4) Pleural adhesions are formed as the result of acute or subacute pleurisy. A dry friction sound is present over a large area for a considerable period. (5) Fibrosis of the lung, followed by retraction and thickening of the pleura; hemoptysis may occur and may prove fatal. Emaciation, dyspnea, and, possibly, pyrexia are present. (6) Pyrexia of a remittent type, due to the absorption of the septic contents of the tubes; or, possibly, rigor and severe general symptoms may be present. Death may be due to pericarditis, septicemia, pyemia, cerebral abscess, nephritis, diffuse bronchitis, or septic bronchopneumonia with necrosis of the consolidated areas.—Ed.]

We see immediately that the pure course of bronchiectasis is represented only by the first form. In all the other cases the bronchiectasis is complicated by other serious conditions, which must obscure the true picture.

Rendu calls attention to a course which is characterized by a pseudo-acute beginning: A healthy appearing young man coughed up suddenly a large amount of pus and died within a year of bronchiectasis. This one case, however, leaves room for doubt.

That fully developed bronchiectases heal only with great difficulty can easily be seen, yet the possibility of an obliteration by the formation of scar tissue should not be disregarded. Such results must, however, be very rare, and are not supported by authentic case-histories.

In some of the cases death may occur quickly from hemoptysis, or an infection from gangrene of the lung or putrid bronchitis may lead to death; the putrid bronchitis may, however, become retrogressive. As rare results may be mentioned, death from pneumothorax, metastatic abscesses, tuberculous infection of the bronchiectatic patient, venous stasis and dropsy, amyloid degeneration; the most frequent manner of death is by the development of bronchiectatic pseudophthisis.

Symptoms.—The *expectoration* does not usually act differently from that in ordinary bronchitis, but in many cases its kind and appearance are characteristic. The secretion has a great tendency to collect and be expelled at intervals in larger quantities. Thus, the sputum may come at once in such large amounts that we believe that an empyema has emptied itself after rupturing into a bronchus. Barth saw 450 c.c., Biermer 640 c.c., and Trojanowski as much as 800 c.c. emptied in twenty-four hours. Generally the expectoration takes place easily, without much cough and without great exertion, yet the opposite is found with emphysema, pleurisy, and stenosis of the larger air-passages. Change of position may be of great importance; lying on one side the patient is quite quiet, but if he turns to the other side, the evacuation begins. Toward the end, when the patients become very weak, the amount of the discharge often decreases with surprising suddenness. [Expectoration may be easy or effected with difficulty, owing to the presence of advanced emphysema or stenosis of the main bronchus, or of marked induration of the tissue around the dilated tubes.—Ed.]

The tolerance of the patient to the abundant and very offensive secretion is worthy of note; complaints are rarely made of nausea and vomiting.

The *sputum* is mostly purulent; it is very yellow in color (if it is not fetid or gangrenous), and has little tenacity. It contains pus-corpuscles, which may be very well preserved, but which also contain many fat droplets. Vacuoles may often be seen in them. Not rarely the sputum forms two layers in the sputum-glass: on the bottom a thick, exclusively purulent layer, containing all kinds of granular material, and over this a thin, slightly turbid, almost serous fluid. It diffuses at times no odor at all, but often an unpleasant, nauseating, sour, stale, or garlicky odor (pseudogangrene); the genuine fetid and decomposing odor I have observed as a sign of a complication (gangrene).

Blood in the discharge is not so very rare; it is generally mingled with it in small amounts. But there may also be quite large amounts of blood in the sputum; death has occurred from hemoptysis. It may result from the erosion of the larger vessels without a trace of tuberculosis. The blood is rarely very red, but is discolored by admixture with the sputum, or the sputum receives the color of meat juice (according to Jaccoud). [Hanot and Gilbert have connected the occurrence of hemoptysis in bronchiectasis with the marked alterations in the blood-vessels which may form in the submucous tissue an extensive cavernous network interspersed with numerous minute aneurysms.—Ed.]

Cough is the rule; it is paroxysmal and is especially frequent in the morning, until the lungs are freed from the masses of pus accumulated during the night. The cough is often especially severe in certain positions and disappears on changing the position. This depends on the position and opening of the bronchi. So long as the discharge remains in the cavity, it produces no cough, and only when it flows out and reaches the relatively healthy mucous membrane, especially when it touches the irritable zone, it excites cough. If the cavity is at the apex, the secretion flows down through an almost vertical bronchus unceasingly and excites an almost continuous cough. [Fox states that the acute bronchiectases of infantile bronchitis are due to cough pressure, rather than to any inspiratory mechanism or to any indirect effect of collateral collapse. As an explanation of the infrequency of bronchiectasis in spite of the great frequency of bronchitis, he alleges that the dilatation is readily recovered from in children, and that in adults chronic bronchitis tends to hypertrophy rather than to weakness of the muscular fibers, in contrast to its action upon the pulmonary parenchyma. Those who are interested in this question of pathogenesis are referred to Allbutt's "System of Medicine," where an elaborate exposition of the various views, including the author's, will be found.—Ed.]

Dyspnea is slight or wholly lacking; but as the disease progresses, it usually increases and may reach a considerable degree. It is then ordinarily due to complications: diffuse bronchitis, emphysema, dilatation of the heart, ascites, and enlargement of the liver from stasis.

The *physical signs* are those of a cavity in the lung.

Deformity of the thorax and marked retraction or drawing in of a circumscribed portion are often to be observed. With this we see rarely and in very severe grades a kyphosis of the vertebral column. The underlying organs are subject to changes of position from the disturbance. Occasionally these signs are concealed or even compensated by an emphysema of the lung. If one lung is especially involved to a marked degree, the other usually develops a compensatory emphysema. In rare, very severe cases, it has been observed that the respiratory movements became weaker or entirely abolished when the corresponding, dilated bronchi were filled for the greater part with secretion. After abundant expectoration the movements began again, to disappear as the secretion gradually accumulated.

Pain is never present as a characteristic of the disease; on the contrary, the absence of pain with the great objective symptoms is noticeable. [Pain in the chest is usually due to pleurisy, and is of importance as showing that the affection is approaching the surface of the lung. In cases originating in an attack of acute pleurisy there will almost certainly be a history of severe pain on the affected side. —Ed.] At times there are in this disease, as in every chronic disease of the lungs, intercurrent pains. Attempts were made earlier to decide on the seat of the bronchiectasis from the complaints of pain in a certain region (Barth), but this was a mistake. They do not belong to the bronchiectasis itself.

Fever is not a symptom of simple bronchiectasis, but indicates complications, depending on infection of the contents of the cavity. Bacteriologic investigation in bronchiectasis is no doubt destined materially to enlarge our knowledge of the subject.

In advanced stages the fever usually presents a hectic type. This depends on the development of masses of cocci, of which we will name only the well-known Streptococcus and Staphylococcus pyogenes, as well as Pneumococcus, and Bacillus coli communis. All these, as well as some rarer forms which Babes found in bronchiectasis, are found in the febrile forms in the various organs of the body, evidently passing into the blood from the cavities. Thiroloix describes a case of bronchiectasis in which Staphylococcus aureus had to be considered as the cause of a secondary endocarditis, of a hepatitis with miliary abscesses, and of a large abscess of the kidney.* Simple septicemia due to infection by Streptococcus pyogenes is a quite frequent occurrence.

A peculiar change in the appearance of the fingers and toes has long attracted attention under the name of *clubbing of the fingers and toes*. It is due to a swelling of the finger by hypertrophy of the terminal phalanx. This hypertrophy is seen first at the ends of the fingers; later it appears also at the ends of the joints, so that the phalangeal, metacarpal, and carpal joints, and even the wrist bones, appear much thickened. The symptoms are just the same in the feet. In this high degree the term clubbing ("drumstick" fingers) is not exactly characteristic; we can therefore use the name given by Marie, "hypertrophic pneumonic osteoarthropathy," which unfortunately is rather unwieldy. The qualifying adjective pneumonic calls attention to the fact that this pathologic condition occurs in diseases of the air-passages. When these become permanent and lead to a lasting cyanosis, the condition described is in truth to a certain degree characteristic of the disease. It belongs, however, not to a specific pathologic condition, but is observed, so far as we now know, just as much in tuberculosis as in chronic bronchitis and bronchiectasis, when the disease has lasted a long time, and has led to chronic cyanosis. This chronic cyanosis seems to be the one essential factor necessary for the development, as there are, for instance, certain diseases of the heart which also cause clubbing of the fingers. The most chronic forms of congenital heart disease, as is well known, especially tend to these formations. Bamberger, moreover, supports the view that the change in bronchiectasis is a quite peculiar form, which is characterized by the painful swelling of the epiphyses. He shows also that the spongiosa as well as the cortical substance of the thickened epiphyses undergoes hypertrophy.

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* *Soc. anatom.*, March 13, 1891.

Concerning the *color of the skin*, Trojanowski gives the following general rule: In the bronchiectatic patient, the color is usually unchanged; marked pallor indicates tuberculosis, and decided cyanosis suggests emphysema.

Position.—This is extremely changeable; many patients lie on their backs, while others generally sit somewhat bent forward. Some of these say that they do it in order not to swallow the sputum (Trojanowski). If one side is attacked by the bronchiectasis with especial severity, the patients prefer to lie on that side, and when they turn to the other side, coughing and profuse expectoration begin.

[*Inspection:* The chest may be emphysematous or there may be retraction of the chest-wall in localized areas and displacement of the cardiac impulse.

Palpation: The vocal fremitus and resonance are increased in cases accompanied by fibrosis.—Ed.]

Percussion gives dulness, but also tympanitic sounds, and in certain places the two alternate, according as secretion or air predominates in the cavity. A tympanitic sound, with change of sound on opening and shutting the mouth, may be discovered oftener than is generally believed, if the ear is held close to the mouth of the patient. Also change of sound with change of position and râles of the cracked pot and metallic type were observed in corresponding cases, according to the same laws which govern their appearance in cavities. Around the place where the signs of a cavity are found dulness may be present; but when the note is resonant, this is important as being especially characteristic, for around tuberculous cavities a dull sound is always present. [Fibrosis around dilated tubes filled with secretion gives rise to an absolutely dull sound on percussion; when they are empty, the note is raised in pitch and may present various degrees of dulness up to a distinctly wooden quality.—Ed.]

Percussion frequently shows a displacement of the neighboring organs and attachment of the same to the lungs, as is easily explained by the retraction of the thorax and frequent occurrence of pleuritic adhesions.

[*Auscultation.*—The respiratory murmur is weakened, or numerous bronchial râles are to be heard, especially large, moist, bubbling râles; systolic murmurs of course also occur in the neighborhood of the heart. The vocal resonance is increased in cases accompanied by fibrosis and bronchophony, and pectoriloquy may be present. Fowler and Godlee lay some stress on the character of the breathing, which is blowing but not cavernous, and the sound may be interrupted both on inspiration and expiration. With sacculated dilatations the breathing may be truly cavernous and gurgling râles may be present. The *veiled puff of Skoda* may often be heard. This is a curious sound occurring at the end of inspiration and giving the impression that a puff of air has suddenly entered a small cavity situated just beneath the ear. When once recognized, it is easily distinguished, and in the authors' experience is more often present in bronchiectasis than in any other condition. The *typical sound* of bronchiectasis is a coarse râle of ringing or metallic quality which may disappear and reappear according to the quantity of secretion in the tubes. Mention is also made of the *systolic râle* produced by the movements of the heart. The case of a man under the writers' care is cited, in which a systolic murmur could be heard at a distance of two feet from the patient when the mouth was open, and its presence led to the suspicion of aneurysm.

Ewart, in Allbutt's "System of Medicine," gives the following points as guides in the *physical examination.* A solitary bronchiectatic lesion is seldom localized at the apex. The supraclavicular area is usually not implicated in any dulness due to bronchiestasis; it is invariably implicated in the apex dulness of phthisis. In phthisis consolidation precedes, excavation follows; in bronchiectasis this is otherwise. Again, the extension of the excavation is peculiar to phthisis (Stokes), while a stationary size belongs to bronchial dilatation (Walshe). The almost daily alternations between a sense of fulness and that of vacuity greatly help the diagnosis of sacculation, as this peculiarity is usually absent or inconstant in excavating phthisis. The site of predilection for bronchial dilatation is the base, but bronchiectasis also favors the middle and lower thirds of the back; it is especially uncommon in the district of the vertical bronchi pointing to the apex. It is unusual in phthisis for multiple excavations to form in the same lung with the intervention of sound pulmonary substance; in multiple bronchiectasis a truly sporadic arrangement is the rule. Unilateral indurative tubercular phthisis invariably excavates and condenses the apex first, even if later it should extend downward. The fibroid change associated with bronchiectasis originates, as a rule, at the base and spreads upward. The displacement of the heart toward the diseased side of the chest in unilateral phthisis follows an oblique direction upward. In unilateral bronchitis the displacement is, practically speaking, always horizontal.

Ewart described the character of the sounds produced in sacculation by the term "*croaking.*" He says it is partly due to the valvular action of the viscid and confluent secretion, and partly to the free communication and continuity subsisting between the sacculations and the corresponding bronchus. The croaking sound is most distinctly produced in sacculations more or less surrounded by spongy tissue. In the fibrotic variety the solid medium through which it is conducted to the ear imparts to it a more metallic character.—Ed.]

In women *disturbances of menstruation*, explicable partly through stasis and partly through cachexia, are not unusual.

Complications.—The most frequent and most dangerous are those which arise from the putrid or gangrenous decomposition of the contents of the cavity; this is often regarded as belonging to the typical picture of the disease. The sputum has such a terribly offensive odor that the glass has to be kept covered constantly; on allowing it to stand, however, this odor diminishes and may entirely disappear. The breath often has a much more offensive odor than the sputum. When the stench from the sputum was so fearful, Bamberger found in it hydrogen sulphid, ammonia, acetic acid, butyric acid, and formic acid.* (Compare Fetid Bronchitis.)

We should also consider the following diseases as being in close relationship with, or as sequels of, bronchiectasis:

Affections of the pleuræ, peribronchitis, bronchopneumonia, lobar pneumonia, hydrops and anasarca from diseases of the kidneys or heart or those of a cachectic nature [amyloid disease, septicemia, and pericarditis (Ewart)].

Emphysema is a frequent and important complication, as the subsequent course of the disease depends upon it; chronic catarrh of the larynx without ulceration; albuminuria; chronic affections of the kidneys; and cirrhosis of the liver are also to be mentioned.

Abscess of the brain, generally on the same side as the bronchiectasis.†

Abscess of the spinal cord (Nothnagel).

Amyloid degenerations (Trojanowski, three cases).

Leichtenstern ‡ mentions a very remarkable complication: A stonecutter had two traction diverticula in the esophagus, and one of the two broke through into a bronchiectatic cavern of the left upper lobe.

Pyopneumothorax, in consequence of the rupture of a cavity.

Rheumatism of different joints, especially of the ankle- and knee-joints, is so frequently noted that Gerhardt § expresses the opinion that it is due to a pus infection. [A case of this character was recently seen by the editor, in which the joints simulated in appearance an acute rheumatoid arthritis. There was high fever.]

Tuberculosis is not a frequent complication, but it is found often enough, in the lungs as well as in other organs (for instance, in the genito-urinary organs; Biermer, 14 cases).

Cancer is an accidental complication (Biermer, Cases 4, 21), but in Case 17, multiple sarcoma of the lung was certainly the predisposing factor, as it caused stenosis.

Gastric ulcer is also mentioned as an accidental complication (Trojanowski, 1 case).

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* *Würzburger med. Zeitschrift*, 1861, vol. II, p. 333.

† Williams, *Lancet*, Dec., 1882; Biermer in *Virchow's Archiv*, XIX; Case 2 (left bronchus, brain abscess on the left, but also pus in the right posterior horn). Cayley, *l. c.*

‡ *Deut. med. Woch.*, 1891, 14, 15.

§ *Deut. Arch. für klin. Med.*, vol. XV.

The majority of bronchiectatic patients suffer from disturbances of the heart, so that this may exert an influence on the course of the disease. In extreme cases the pleural thickening may extend to the pericardium, and this may become adherent to the heart. Hypertrophy and dilatation of the right ventricle are much more common. True valvular lesions are rare.

Diagnosis.—After the detailed description that has been given, it is only necessary to mention that it is possible to confound the disease with all its complications, and that it is most frequently confounded with simple bronchitis.

The differential diagnosis from tuberculosis, which was formerly so difficult and called for so many and such uncertain observations, has now become one of the surest that we have, thanks to the discovery of the tubercle bacillus; although the coexistence of tuberculosis and bronchiectasis cannot be determined by means of this investigation. But no more do the older auxiliary factors (age of the patient, situation of the cavity, general habit) assist us in arriving at a diagnosis; a decision can be reached only by a knowledge of the entire history of the case. As a fundamental principle we may accept the axiom that tuberculosis may occur as a complication of bronchiectasis, but not the reverse, which is practically correct.

Confusion with gangrene of the lung, putrid bronchitis, interstitial pneumonia, and pneumothorax is less easy to avoid on account of the fact that these things so often occur together. Besides, we cannot, for the diagnosis of bronchiectasis, insist on the demonstration of large air-containing cavities, since the cavities are often enough filled with pus and mucus instead of air, and are frequently not large enough to affect auscultation and percussion in any characteristic manner.

The cases thus divide themselves into two groups: First, those which present cavernous symptoms and in which we must guard against confusion with tuberculosis, and those rare diseases which present the same symptoms, such as abscesses, gangrene, pneumothorax; and, second, those which show no signs of cavity; in the latter a more accurate diagnosis than "bronchitis," "putrid bronchitis," or "chronic pneumonia" can only be inferred.

[According to Fowler and Godlee, the diagnosis from chronic bronchitis with emphysema depends chiefly upon the character of the expectoration and the peculiar mode of its evacuation. Persistent fetor points strongly to bronchiectasis. If the breathing-sounds are of the wavy, bronchial quality, and accompanied by coarse bubbling or ringing metallic râles, and particularly by the "veiled puff," it is very probable that the tubes are dilated.

To decide the question of operation it often becomes important to differentiate between bronchiectasis and a limited empyema with an opening into the lung. In empyema an operation is decidedly indicated, whereas in bronchiectasis it is seldom advisable, except in those rare cases in which a single large bronchiectatic cavity is present in the lower lobe, when an operation is admissible and has been attended with success.—Ed.]

Treatment.—Recovery from bronchiectasis has been at times the result of an obliterative process by the growth of connective tissue; but such a spontaneous cure can be expected only in rare and especially favorable cases, like that of Bamberger, for example, in which recovery occurred after the cavity had ruptured into the pleura.

However, we believe that in time the effort to discover and fulfil the conditions necessary for recovery will be successful. There is no doubt that a fundamental condition is to keep the contents of the cavity and bronchi aseptic, the only means of accomplishing which is, so far as we now know, to keep the patient in the purest possible air. This therapeutic indication can be in part satisfied by the internal administration of creosote and turpentine.* We may also provide for the inhalation of creosote solutions and turpentine vapors; but these laborious and circumstantial methods will accomplish relatively little, and internal medication will have to be regarded as the superior method. Grainger Stewart asserts that injections into the trachea have given him the best results. He recommends †:

Menthol	10 parts
Guaiacol	2 "
Olive oil	88 "

Inject into the trachea twice a day.

Not drugs, but a suitable place of abode and appropriate occupation, are the most important factors in the treatment of bronchiectasis; the patient must, above all things, avoid the haunts of men, for crowds are the hotbeds where the injuries most to be dreaded multiply. The greatest care must be exercised to avoid any new inflammatory process, even a mild attack of bronchitis; and this indication, together with the foregoing one, make up the fundamental principles.

The symptomatic use of expectorants, such as ipecac, senega, and apomorphin, may be useful at times in these cases during the stage when there is a certain stagnation of the secretion due to tolerance of the bronchi, and it is desirable to stimulate evacuation of the secretion. But I always object to the long-continued use of these drugs.

[A practical point of some importance consists in having the patient lower the head, either over the edge of the bed, or while standing, to allow the accumulated secretion to gravitate out of the sacculations and into the receiver. Its regular employment should be suggested whenever no contraindications exist. Ewart ‡ recommends raising the foot of the bed twelve or fourteen inches. Great relief from the paroxysms of cough and gushes of pus ensue. In one case the fever lessened and the expectoration was reduced.—Ed.]

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* Tolu and thymol are also used in a similar way, but give no better results. Recently myrtol has been very highly recommended in doses up to 6.0 grams (ʒ iss) a day.

† *Brit. Med. Jour.*, 1893, I.

‡ *London Lancet*, July 18, 1901.

Some time ago the attempt was made to treat the bronchiectatic area directly with antiseptic applications. Weak solutions of carbolic acid and other disinfecting fluids were injected into the lungs by means of a Pravaz syringe in the hope of reaching the seat of the cavity or cavities. A consideration of the extreme complexity of the conditions that confront the therapeutist in these cases, and a study of the postmortem appearance of such a lung with its winding and intertwining cavities filled with pus, out of all proportion to one or even ten syringefuls of a fluid that can only act for an instant, will enable one to form a true estimate of the value of local disinfection. Were it possible to diagnose a circumscribed cavity, it might be rational to attempt cure in this way.

[One of the recent methods of treatment is the *inhalation of creosote vapor*, as suggested by Dr. Arnold Chaplin. The method is as follows: A small room should be set apart for the purpose and should be cleared of furniture, except such articles as wooden chairs and a table. The patient should put on, over his clothes, a garment with sleeves, shaped something like a smock-frock, to prevent the smell of the creosote vapor from penetrating the clothing. The eyes should be protected by well-fitting goggles or by a mask made of adhesive strips with two watch-glasses corresponding in position with the eyes and tied with strings at the back of the head. Plugs of cotton should be inserted in the nostrils. Ordinary commerical creosote is poured into a metal saucer on an iron tripod and the saucer heated with a spirit-lamp placed beneath it. As the creosote is heated, dense clouds of vapor arise and quickly fill the room. The effect on the patient is to produce violent cough attended with profuse expectoration, nearly two-thirds of the daily quantity being usually ejected during the bath; vomiting may follow the cough. The process is not a pleasant one, but it is remarkable how readily some patients become accustomed to it. They usually state that the breathing is much freer after the bath and that they enjoy a much longer interval of almost complete freedom from cough and expectoration. At first the bath may be given on alternate days from fifteen to twenty minutes; but when the patient has become accustomed to the treatment, the bath should be given daily and the duration of it gradually increased up to an hour or an hour and a half. The treatment should be persevered with for long periods, a bath being taken daily for three months or more. For a full discussion of this method, with clinical notes of a number of cases, the reader is referred to Fowler and Godlee, "The Diseases of the Lungs.". The authors come to the following conclusion with regard to the creosote vapor bath treatment: (1) It presents no practical difficulties and is unattended by any unfavorable results. The benefit to be obtained in bronchiectasis is far greater than that derived from any other form of treatment. (2) In exceptional cases a condition almost amounting to cure may follow the prolonged use of creosote vapor baths. (3) As a rule, the fetor of the sputa is modified, and not infrequently the odor disappears altogether, but in some cases it is unaffected. (4) The quantity of the sputa is not always, or even as a rule, diminished. It varies much from time to time and may be increased. (5) The treatment requires to be continued for a long period, possibly six months or more; in some cases its continuous use is advisable.

Another method consists in *hypodermic injections of sterilized oil of guaiacol or creosote.* The dose is 30 minims of a solution of the strength of 1 : 4.

In regard to the *inhalation of oxygen,* it theoretically fulfils a double purpose: that of aiding respiration, and of disinfecting the air-tubes. Actually, however, it has proved of little value, perhaps because of its tendency to diminish rather than to increase the activity of the respiratory movements.

Poore is the originator of a method based upon the penetrating properties of some of the volatile constituents of *garlic,* and upon their stimulating and antiseptic, as well as odoriferous virtues. Garlic probably acts as a general tonic as well as a local stimulant. Its local effect is produced at the surface of the mucous membrane by exhalation, but the fact that the smell of garlic is also given off by the skin suggests that the constitutional influence of the drug may be wide-spread and important. Poore reports that the original fetor of the expectoration in the cases treated with garlic was replaced by a pungent smell reminding one of that of syringa. The discharge was greatly diminished and a remarkable improvement took place in the health, in the strength, and in the weight of the patients. The treatment is generally well borne, and if the remedy be taken after meals, the patients generally submit without much inconvenience. A clove of garlic is chopped up and mixed with beef-tea, or preferably inclosed in gelatin capsules. As much as 30 grains of chopped garlic have been administered in capsules, eight times a day. Poore suggests that sulphid of allyl is probably the remedial agent. The *oil of allyl* may be prescribed in capsules containing ½ a minim to begin with, and the dose gradually increased. Good results have been obtained by this method, not only in bronchiectasis, but also in phthisis.

In the tuberculous cases Allbutt regards the use of intratracheal injections with grave suspicion, on account of the possible dissemination of the infection from the upper into the lower lobes.

In view of the growing popularity of the *spa treatment,* a note in regard to the climatic and crounotherapeutic possibilities in bronchiectasis may not be out of place. In England Thanet, Folkstone, Easton, and Brighton are recommended. The climate to be sought is a dry, or even a cold atmosphere, such as that of the Alpine winter, with plenty of outdoor life and physical exercise. Accordingly patients in the United States may, if no contraindications exist, be sent to the high Western plateaus, or, if desired, to the more hospitable southern shore of California. Among medicinal springs the sulphurous thermal waters enjoy deserved reputation in the treatment of this affection. Harrowgate, Moffatt, Aix-les-Bains, Eauxchaudes, Cauterets, and Bagnères-de-Luchon; and, in the United States, Warm Springs, Virginia; Richfield Springs, New York; Hot Sulphur Springs, Colorado; and Gilroy Hot Springs, California. Patients unable to leave home may find a substitute in tonic baths combined with the internal administration for recurring periods of some preparation of sulphur. Warm sea-water baths may be of considerable value, and in some cases the stronger stimulation obtained from the artificial Nauheim baths may be desirable. A tepid bath terminated by cool or cold effusion, and followed with strong friction of the surface, may be recommended among hydriatric measures.—Ed.]

The futility of local applications has led the surgeons to go still further; they have punctured the cavities with a trocar and inserted a drain through the opening *; and, finally, they have searched for them with a knife and opened them. When we think how uncertain the diagnosis is, how rarely a single cavity is present, what dangerous hemorrhages and other accidents have accompanied the operations on the lungs, we will not place too much reliance on this procedure; however, it cannot be entirely disregarded, as the advances in operative technic promise better and more reliable results than have been gained hitherto. For the present, under favorable surroundings, the chances for the patient are better without than with operation. In special cases operation may be considered, when a circumscribed cavity, the location of which can be established with the help of an exploratory puncture may be reasonably assumed, and when the obliteration of the pleural cavity at the site of operation has been assured.

The results of pulmonary surgery in bronchiectasis, as they appear at the present time, may be gathered from a study of the following cases, which I was able to collect:

1. Cases resulting in recovery or improvement:

Mosler and Hüter, 1875.
Lauenstein: "Chirurg. Centralblatt," 1884, No. 18.
Rochelt: "Wiener med. Presse," 1886, No. 39.
Albert: "Wiener med. Presse," 1886, No. 39.
Godlee: "Brit. Med. Jour.," March, 1886, p. 590, two cases.
Léonville: "Revue méd. de la Suisse romaine," v, 1886, two cases.
Stewart: "Brit. Med. Jour.," 1887, p. 179.
Hofmockel: "Wiener med. Presse," 1892.
Hofmockel: ibid., 1893, No. 18.

2. Cases resulting in death:

Mosler and Hüter, 1873.
Douglas: "Lancet," ii, 1879.
Koch: "Centralblatt für Chirurgie," 1882, p. 693, two cases.
Mosler and Vogt, 1882.

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* Th. Williams.

Rendu and Segond: "Rendu leçons de cliniques méd.," I, p. 169. (Operation with good results, but death from collapse on the third day.)
Williams: "Lancet," 1882. (Death from abscess of the brain.)
Brookhouse: "Lancet," 1888. (Bronchus opened on account of symptoms of septicemia. Unsuccessful. Death.)
Mackey: "Brit. Med. Jour.," Oct., 1888, p. 765. (Bronchus after pleuropneumonia in a man twenty years of age. The operation was followed by temporary relief, then death from septic pneumonia.)
Finlay: "Brit. Med. Jour.," Oct., 1888, p. 807. (Severe bronchitis in a ten-year-old boy, resulting from a splinter of bone. Operation. Death after fourteen days.)
Krecke: "Münchener med. Wochenschr.," 1891.
Biondi: "Chirurg. Centralblatt," 1894.

I have found, then, 11 cases of recovery, as opposed to 12 cases of death. The work of Roswell Park,* which is said to contain 23 cases, was not accessible.

Hofmockel has given an exhaustive discussion, with a collection of 80 cases operated on account of abscesses, gangrene, or bronchiectasis. The best results were secured by the operation for abscesses; the worst by that for bronchiectasis. [See Abscess and Gangrene of the Lungs for further discussion of the surgical treatment of this affection. —Ed.]

VICARIOUS BRONCHIECTASIS.

When a portion of a lung has become inaccessible to the air and its volume is reduced, the traction exerted on the adjoining tissues during inspiration will cause them to make an attempt to fill the space. The powerful pull of the muscles which dilate the thorax will not be affected by the resistance of smaller areas, although in the case of the larger ones the excursions of the thorax will, of course, be somewhat restricted. The pull which acts on the lungs being unable to expand the impermeable portions, will expand the other portions; according to the position and circumstances, this may affect the alveoli or the bronchi, and thus arise vicarious emphysema and vicarious bronchiectasis, hence the not infrequent association of the two conditions.

There may, therefore, be bronchopneumonic masses of every kind, atelectases, infarcts, and cicatricial retractions. The anatomic conditions are very clear in the well-marked cases: the mucous membrane seems thinned like a serous membrane. We also find occasionally a hint of the distinction between the atrophic and the hypertrophic form of bronchiectasis, and under the influence of the contraction of scar tissue the atrophic form may undoubtedly develop from the hypertrophic; the important point is that no inflammatory processes play any part in this form of the disease; it is a true emphysema of the bronchi. Rokitansky in describing certain emphysematous lungs states that the bronchial tubes were everywhere dilated and beset with lax, thin-walled sacs, which had developed through dilatation of the fine branches going from them.† These cases we must certainly consider as vicarious.

**Annals of Surgery*, 1887. † "Lehrbuch der path. Anat.," III, 54, Wien, 1861.

Clinically vicarious bronchiectasis has very little importance; the dilatation does not become great enough to form demonstrable cavities. Besides the diseases which have an assignable cause and which still predominate in the pathologic picture, we find a disease which is in the main a bronchitis, but which will excite suspicion on account of its blennorrheic sputum.

The possibility that the bronchi may also become ulcerated and that they may furnish the basis for the development of inflammatory bronchiectases is, of course, not excluded. But this also is to be regarded as rare. We know that neither tuberculosis nor emphysema especially predispose to inflammatory bronchiectasis, and that these are the two main diseases that favor the vicarious type in the diffuse form. The other causative diseases lead to the formation of circumscribed masses.

When conditions are found which favor stagnation of the secretion, quite peculiar masses of smaller inflammatory bronchiectatic areas, or a single larger one, may develop, which remain perfectly inexplicable unless we consider this relationship.

CONGENITAL BRONCHIECTASIS.

Under the name of congenital bronchiectasis an affection of the bronchi has been described which is found in the new-born. The lungs, or usually one alone, are filled with cysts which contain a serous fluid. The disease is considered as a manifestation of hereditary syphilis.

Grawitz * found the right lower lobe converted into a multilocular, flaccid pouch, which did not communicate with the bronchi. This pouch was filled with a thin fluid containing nuclei and ciliated cells; the walls were lined with several layers of cuboidal epithelium, the uppermost cells of which were ciliated. Further investigation indicated that it might have been a hydropic distention of a main bronchus, which had led to cyst formation. A system of cavities was shown, which opened together into a common middle space and were separated from each other by thin dividing walls. From the largest arose secondary and tertiary party walls, so that numerous recesses were formed. A small boundary zone of lung substance formed the transition from the cyst to the rest of the healthy lung. This boundary zone, which appeared almost emphysematous in the recent lung, consisted of embryonal lung tissue.

Kessler (Meyer), whose case Grawitz cited, observed a girl who was healthy until the fifth month of her life, and then began to suffer from attacks of asphyxia, aggravated by lying on the right side and relieved by lying on the left. A permanent cyanosis developed. The right lung was found somewhat emphysematous at the borders; the upper part of the left formed a wide fibrous sac connected with the

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* *Virchow's Archiv,* vol. LXXXIII.

root of the lung by a small amount of normal lung tissue. Numerous bronchial branches opened into the sac.

Meyer (*ibid.*) found in a five to six months' fetus a very similar condition; and, finally, Fraenkel mentions a similar finding in a twenty-five-year-old gold-worker, who died with the symptoms of severe asthmatic trouble. Grawitz considers it highly probable that this was also a congenital case.

This group of cases is compared by Grawitz with others which are likewise to be regarded as developing during fetal life. But there are very many cysts; they are located in the bronchi of the third and fourth order, are often arranged in rows one behind the other, sometimes opening outward and sometimes entirely closed. A shrunken connective tissue or a slaty induration is generally wanting between the cysts; only one lung is attacked, and, like the affected portions of the preceding group, is characterized by an entire absence of pigment. The individuals of this group also had goiter.

[E. Neisser * reports four cases of *unilateral atrophy of the lung*, probably connected with congenital bronchiectasis. The cases resemble each other in their main features. The patients had all suffered from cough, dyspnea, attacks of fever, with foul-smelling or bloody sputum, since earliest childhood. These attacks were intermittent, and in the interval the patient was able to attend to his work. The cases showed a marked atrophy of the left lung, with dislocation of the heart toward the left, a high position of the diaphragm, and an enormous compensatory hypertrophy of the right lung, well shown by means of the Röntgen rays. In these cases there was no deformity of the affected side of the thorax, no "rétrécissement de la poitrine," described by Laennec as one of the cardinal symptoms of an acquired unilateral atrophy of the lung. The author reviews thoroughly the literature of the cases of unilateral atrophy of the lung without deformity of the thorax. He thinks that those factors which are active in the causation of deformity of the thorax in cases of acquired atrophy of the lung may be disregarded in cases of atrophy of the lung which are either congenital or date from earliest infancy. Thus, he cites cases of congenital or early acquired atrophy in which pleural adhesions were proved to be at one time present, but were later confined to the posterior and lateral chest-walls, while the anterior wall was free to grow. After a critical review of the few reported cases of atrophy of the lung in adults, without deformity of the thorax, the author,—though he admits that in case of a very slow development of lung atrophy in an adult, compensatory hypertrophy without deformity of the chest may occur,—is inclined to the belief that all cases of atrophy of the lung in adults with normal chests may be traced to a congenital or very early acquired lesion. The writer goes over the reported cases of congenital bronchiectasis with unilateral atrophy, and believes that his cases belong to this class. Two of his cases occurred in father and son, and for this as well as for other reasons the author believes that heredity has something to do with this affection.—Ed.]

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* *Zeitschrift für klin. Med.*, XLII, 1901, p. 88.

Grawitz names the second form the telangiectatic form, because only the smaller bronchi are attacked, in contrast to the first form, which he would call "universal ectasia"; in the latter there is a large common cavity, with numerous recesses.

[Ewart speaks of *dermoid growth* within a bronchus as among the causes of congenital bronchiectasis, and describes a specimen in the Museum of St. George's Hospital.—Ed.]

THE ATELECTATIC BRONCHIECTASIS OF HELLER.

I will mention here the cases of atelectatic bronchiectasis of Heller. They are distinguished from those of Grawitz by the preservation of portions of fetal lung tissue, by a surprising growth of cartilage in the walls of the bronchi, and by the fact that they contain no ciliated epithelium, the epithelium being squamous; but, on the other hand, they present very marked similarities, and the question always arises whether some of the cases of Grawitz, if they were met in older people, could not be regarded as such atelectatic forms. In recent times more attention is being paid to these atelectatic cases; Herxheimer * mentions three cases, affecting individuals of forty-eight, forty-nine, and sixty-five years respectively. Here, however, the portions which were macroscopically free from pigment showed on microscopic examination finely granular pigment; in one case the hypertrophy of the bronchial cartilage, especially mentioned by Heller, was not present.

In the interesting case of Francke † some pigment was found in the bronchiectatic portions, but appeared quite different from that in the healthy portions of the lungs. This case was further characterized by a remarkable deformity of the thorax,—opposite the lower lobe of the lung, which had been attacked by the atelectatic bronchiectasis, the thorax was constricted in a peculiar manner,—and also by the fact that the foramen ovale had remained open. Francke also mentions Gairdner's case of a man sixty-six years old. In this case the lower lobe of the left lung was found beset by bronchiectatic cavities and was entirely free from pigment. The tissue appeared neither contracted by scar tissue nor changed by interstitial inflammation, and, aside from the absence of air and pigment, had the characteristics of normal lung tissue, so that it was necessary to think of congenital atelectasis.

[BRONCHIECTASIS IN CHILDREN.

The term "bronchiolectasis" has been applied to a condition in which the finest ramifications of the tubes undergo dilatation.

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* *Breslauer ärztliche Zeitschrift*, IX, 1887 † *Deut. Arch. für klin. Med.*, vol. LII.

The affection is found chiefly in syphilitic or rickety children, attacked with bronchitis, which may be either primary, or secondary to some acute specific disease, such as measles, whooping-cough, or diphtheria. The pathologic lesions which specially characterize these cases are acute peribronchitis, dilatation of the bronchioles throughout extensive areas of the lungs, the presence of innumerable small cavities which give the lungs a "worm-eaten" or "honeycombed" appearance, and the presence on the surface of the lungs of small vesicles containing air. In most cases no tubercles are found, but the changes may occur in association with miliary tuberculosis.

The exact *pathology* of the condition known as "honeycomb lung" is not yet determined. By some observers it is doubted whether—at any rate in the tubercular cases—the cylindric dilatations are really dilated bronchioles, and it is certainly rarely possible to demonstrate the presence of any structure belonging to the bronchiole in the cellular exudate forming the walls of the dilatations. In some cases the larger cavities appear to be formed by the breaking-down of septa intervening between neighboring spaces.

Symptoms.—The cough tends to be paroxysmal and may closely simulate whooping-cough. The breath will probably not be fetid, and expectoration may be absent, because it is swallowed, but during the act of vomiting, which may accompany the cough, large quantities of pus may be expelled. Fever is not necessarily present, but during some period of the disease a high temperature (103° to 105° F.) is generally observed. Toward the close, when cyanosis is present, the temperature is generally subnormal. In subacute and chronic cases clubbing of the fingers is often observed. The general condition of the patient may remain fairly good and may be much less unfavorable than would be expected from the extent of the disease within the lungs.

The *physical signs* do not differ materially from those already described. In acute cases tubular breathing may be absent and crepitant râles may be audible over extensive areas. The special feature of the adventitious sounds is their variability from day to day, both in number and in quality.

The *diagnosis* between bronchiectasis and tuberculosis is rendered more difficult in children on account of the absence of expectoration, but marked variability in the signs present from day to day is characteristic of bronchial dilatation. In bronchiectasis there is often a decided want of proportion between the extent of the disease, as evidenced by the results of physical examination, and the general condition of the patient; the latter, indeed, may improve while the disease is extending—an event rarely observed in tuberculosis. The presence of glandular enlargement elsewhere is in favor of a diagnosis of tubercle.

Prognosis.—While recovery is rare and the condition usually passes into the chronic stage, even then, although permanent, the disease is not necessarily progressive, provided the patient is placed under favorable conditions.

Treatment.—Vapor baths have been used in older children, from six years upward, and the method has been attended with the same good results as in adults. Cod-liver oil and tonic remedies, and, when there are signs of deficient aeration of the blood, inhalations of oxygen are advised.—Ed.]

BRONCHOSTENOSIS.

The older literature of this subject is given by Riegel. For the more recent, which is referred to here, see especially under Syphilis of the Bronchi, Tumors of the Bronchi, and Diseases of the Bronchial Glands.

Stenosis of the larger bronchi—the only ones that will be discussed here—may develop from the most varied diseases, and so we shall consider it only in its etiologic and symptomatic aspect as a symptom of great practical importance.

Etiology.—Bronchial stenosis is naturally divided into the affections arising from intrabronchial causes, those from extrabronchial causes, and diseases of the bronchial wall itself.

Intrabronchial causes: (1) Foreign bodies (compare under that heading); (2) malignant tumors which grow into the bronchus.

Case of Powel. ("Path. Soc. Trans.," vol. xxx, p. 249.) Tumor of the posterior mediastinum, which had grown into the right bronchus and had finally entirely occluded it.

Extrabronchial causes bring about compression stenoses:

(*a*) Through diseases of the lymph glands (see under these).

[See also "Bronchial Obstruction by Suppurating Bronchial Gland." *—Ed.]

(*b*) Through aortic aneurysm. Aneurysm of the ascending aorta usually compresses the right bronchus; that of the arch and, especially, of the descending portion compresses the left bronchus.

Landgraf, 1887. ("Berliner klin. Wochenschr.") Aneurysm of the descending aorta which pressed on the left main bronchus.

O. Fräntzel, 1891. ("Charité-Annalen," xvi.) Aneurysm which pressed on the left main bronchus.

Habershon. A similar case. "(Guy's Hospital Rep.," Ser. iii, vol. ii, 1856.)

Irvine. Aneurysm of the aorta which had so pressed on the left bronchus that it had caused perforation. ("Trans. Path. Soc. of London," vol. xxviii, p. 63.) The case is more one of compression than of perforation. The difference between the two lungs is very marked: the one emphysematous with open bronchus, the other with narrowed bronchus, riddled like a sieve by many small cavities.

Irvine. ("Trans. Path. Soc. of London," vol. xxix, p. 36, 1878.) Aneurysm of the ascending aorta compressing the left bronchus. The inner surface was ulcerated. The left lung consisted of a mass of cavities, filled with a reddish, friable material; interstitial pneumonia and bronchiectasis. The right lung was simply emphysematous.

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* J. N. Hall, *Am. Jour. Med. Science*, Aug., 1899.

Irvine. ("Trans. Path. Soc. of London," vol. XXX, p. 239.) (1) Compression of the ascending portion of the arch. The pressure had caused two ulcerated openings in the bronchus. A portion of the corresponding lung was filled with small cavities (honeycombed), another portion contained cheesy masses and gelatinous infiltrates. The pleura was greatly thickened and adherent. (2) Aneurysm with perforation of the left bronchus. The corresponding lung was markedly bronchiectatic. (3) An aortic aneurysm compressing the left bronchus. The left lung was emphysematous and showed some scattered pneumonic foci.

Kidd. ("Trans. Path. Soc.," vol. XXXV, p. 118.) Aneurysm at the beginning of the ascending aorta, the pressure of which affected mainly the left bronchus. The left lung was filled with cavities of varying sizes and contained thick, bloody, and offensive masses. There was no trace of tubercle.

(*c*) Mediastinitis and mediastinal tumors.

Bronchostenosis from an adenoma. Müller. (Dissertation, Halle, 1882.) Secondary bronchiectasis.

Stenosis of the right bronchus, due to carcinoma of the mediastinum. Secondary bronchiectasis. Williams. ("Trans. Path. Soc.," vol. XXIV.)

Stenosis of the right bronchus from a tumor which had grown between the bronchial trunk and the trachea. Bronchial glands enlarged. Dickinson. ("Trans. Path. Soc.," vol. XXIV.) The author considers the tumor a lymphadenoma. These tumors do not, however, usually cause stenosis.

Narrowing of the left bronchus by carcinoma of the bronchial glands. Powell. ("Trans. Path. Soc.," vol. XIX, p. 76.)

Narrowing of the right bronchus by carcinoma of the anterior mediastinal glands. Death from hemoptysis. Moxon. ("Trans. Path. Soc.," vol. XIX, p. 64.)

Lediard. ("Trans. Path. Soc.," vol. XXXI, p. 309.) A lymphoma of the mediastinum had grown into the right lung and so crowded the right bronchus that the lumen was almost obliterated.

West. ("Trans. Path. Soc.," vol. XXXVII, 1886, p. 141.) Medullary carcinoma in the mediastinum compressing the left bronchus. The lung was not more than an eighth of its normal size and was covered by a thickened pleura (5 cm.). In addition, there was a large amount of fluid in the left pleural sac. Both pulmonary arteries were obstructed.

(*d*) Pericarditis, hydropericardium and dilatation of the left auricle.

King. ("Guy's Hospital Rep.," 1838, Ser. I, vol. III, p. 175.) Two cases with mitral insufficiency: one with synechia and mitral stenosis, and one with cyanosis, patency of the foramen ovale, and insufficiency of the valves of both ventricles. Aside from the left bronchus, which was considerably flattened, the bifurcation, and even the beginning of the right bronchus, seemed somewhat compressed.

Friedreich. ("Virchow's Spec. Path. und Ther.," vol. V, 2, p. 236.)

Taylor. ("Trans. Path. Soc. of London," vol. XI.) Mitral stenosis and hydropericardium.

(*e*) Abscesses of the vertebral column, sternum, and clavicle.

Demme. ("Würzburger med. Zeitschr.," vols. II und III.)

(*f*) Tumors of the lungs.

Bronchostenosis from a sarcoma of the lungs. Müthler. (Dissertation, Berlin, 1873.) Secondary bronchiectases.

(*g*) Tumors of the esophagus.

These tumors perforate the bronchi, but rarely cause stenosis. Zucker and Ziemssen have such a case in "Oesophaguskrankheiten," p. 176. Carcinoma of the upper and lower portions of the esophagus, the two separated by a healthy portion, 4 cm. long. Extensive infiltration of the tracheal lymph-glands; trachea infiltrated with carcinoma, as also the entrance of the left bronchus and the left bronchus itself.

It is evident that the stenosis, according to the kind of pressure, may vary greatly in form and extent. Compression stenoses exerting pressure on a circumscribed spot usually lead to an inflammatory swelling of the inner surface of the bronchus, which results in ulceration and may lead to perforation (compare Perforation). The proximal portion of the bronchus regularly becomes dilated, sometimes to a high degree; of this we have already spoken under Bronchiectasis.*

On the distal side of the compression also a marked dilatation is not so very rarely observed. The lung tissue on the proximal side generally becomes emphysematous, owing, probably, to the development of paralysis of the bronchial muscles, obstruction to the outflow, and damming back of the secretion.

Stenosis due to disease of the bronchial wall itself occurs in the following conditions:

1. (*a*) Syphilitic ulcers with scar formation; (*b*) syphilitic peribronchitis (compare Syphilis of the Bronchi).

2. Other ulcers (?). For this there is no authentic material in the literature, although *à priori* it would not be impossible that a tuberculous or traumatic ulcer should have such a result.

3. Tumors (compare these).

4. Simple inflammatory thickening. These cases are found only in the older literature and are very doubtful.† They were characterized by the fact that no bronchiectatic areas had formed behind the stenosis. Chronic blennorrhea of the nasal, laryngeal, and tracheal mucous membranes may extend to the bronchial mucosa, but the disease of the latter, in comparison with that of the upper air-passages, is of very minor importance.

5. Perichondritic processes; and

6. Abscess formations and necrosis of the cartilage. Their etiologic significance is not confirmed by any case-histories.

7. Rindfleisch asserts ‡ that a circular narrowing of the large bronchi at the point of bifurcation and at the points where the secondary bronchi are given off occurs in emphysema. The bronchial wall appears thickened, the cartilages seem to be pushed together and their edges overlap in places. He concludes from this that there is a shortening of the bronchi. This description is in remarkable contradiction to those otherwise known to me, and seems not to have been made the subject of later investigation.

8. Gerhardt has described a very peculiar case of *bronchostenosis ecchondrotica*.§ A man had several times suffered from severe

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* A careful analysis of cases that form an exception to this rule, with a view to determining the factor on which the exception depends, would be of the greatest value. In one of the cases reported by Kopp (*loc. cit.*, p. 312) it is expressly stated that: the left main bronchus is narrowed to such a degree that an ordinary lead-pencil can barely be introduced; at its entrance into the lung tissue it regains its normal caliber.

† Andral, *Clinique méd.*, vol. III, Paris, 1840, pp. 176, 178.

‡ "Sitzungsbericht der physiol.-med. Gesellschaft zu Würzburg," 1886.

§ *Jenaer Zeitschrift für Medicin und Naturwissenschaft*, vol. III, p. 134.

attacks of a bronchitic nature. He died of typhoid fever. In the left main bronchus, and in numerous larger and smaller branches, the cartilaginous rings were found considerably thickened and covered on their inner surface with a layer of bone substance. In places dendritic exostoses, warty and granular growths of cartilage, were present. Some of the cartilaginous rings had a thickness of 4 to 6 mm. The muscles and elastic fibers were arranged in the form of a network, leaving spaces into which the exostoses projected. The mucous membrane itself was thinned and showed none of the above-mentioned bony and cartilaginous growths. The lumen of many bronchi was constricted, but, on the other hand, cylindric dilatations were found in the small bronchi.

Course.—If a sudden closure of one main bronchus occurs, we find a high degree of dyspnea; life is much endangered. Marked disturbances of circulation favor the development of edema of the lung on the side which remains permeable, and thus death may occur very quickly. If, on the other hand, the patient survives this shock, or closure develops slowly, there results only a constriction, the patient's life is not endangered, and even the dyspnea is surprisingly slight and may not be noticed by the ordinary, inattentive observer. The patients complain, however, that active movements,—rapid walking, climbing stairs, and the like,—are very hard for them and make them short of breath.

When the constriction exists for a longer time, conditions develop in the affected lung, a knowledge of which was necessary for the understanding of bronchiectasis, and which were, therefore, given under that head: atelectasis, emphysema, bronchiectasis, and bronchopneumonia (compare page 190). The subsequent course depends on the development of sequels. Here I will mention only the direct consequences of constriction or occlusion of a main branch. The symptoms of possible sequels will not be considered here, and the reader is referred to the above-mentioned sections.

Symptoms.—As soon as the obstruction becomes so great that it is an essential hindrance to the passage of air, disturbance of the respiration results. But this is quite the same as that in tracheal stenosis, in which a bilateral bronchial stenosis develops, which is very rare.* If only one bronchus is obstructed, the air entering through the other is sufficient to satisfy the respiratory needs so long as the other lung is essentially healthy. The number of respirations is then somewhat increased, but the type of respiration is not changed. If the main bronchus is narrowed, the respiratory murmur on the affected side is weakened or abolished; the vocal fremitus and the thoracic excursions are affected in a similar way; the percussion note is resonant, if no complications have developed in the lung. The inspiratory retraction of the intercostal spaces is seen to a surprising and characteristic extent. The other side, on the contrary, bulges, its respiratory excursions are of considerable extent, and the diaphragm is lower than normal.

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* As in the case of Favroud, who observed a bilateral stenosis from syphilis in a sixty-three-year-old woman. In the case of Immermann (*Deut. Arch. für klin. Med.*, vol. v), the first bronchial branches on both sides showed several marked constrictions and angular bends, which caused a narrowing of a portion of the lumen to mere cleft-like openings. There was a thick, hard mass in the hilus of the lung which had also constricted the pulmonary arteries. The respiration was accelerated and markedly dyspneic, but nothing definite was said about the type.

We often hear, over the point corresponding to the stenosis, a peculiar whirring sound which can even be felt as a thrill. This may be communicated to the speech, and the voice takes on a peculiar bleating tone (Punch's voice). If the stenosis affects one of the principal branches of the main bronchus, this may be revealed by the weakened breathing in a corresponding extent.

Pain is usually not present to any marked degree; on the other hand, there is often a feeling of pressure, a sore feeling on one side in the depths of the chest, very definitely localized. According to the etiologic factors, however, the pain may be very severe, especially so in carcinoma.

TRACTION DIVERTICULA.

Traction diverticula have sometimes been observed in the large bronchi, as by Immermann.* In this case there was an extensive formation of wheals at the hilum of the lung. The mucous membrane of the right bronchus between the first and second cartilaginous rings was pushed out like a diverticulum, and drawn toward an extremely narrowed branch of the pulmonary artery; a narrow, cleft-like opening led into a pocket nearly 1 cm. in depth. Among the interesting preparations of Tiedemann,† the tenth shows between the third and fourth cartilages of the left bronchus a small diverticulum 3 mm. in depth. At the outer side of the bronchial tube, embedded in a mass of scar tissue, was found the remains of a calcified, shrunken bronchial gland.

Traction diverticula of the bronchi have no clinical importance.

* *Deut. Arch. für klin. Med.*, 1869, p. 235.

† *Deut. Arch. für klin. Med.*, 1875, vol. xv.

PERFORATION OF THE BRONCHI.

LITERATURE.

Vigla: "Schmidt's Jahrbücher," vol. LXXXVIII, p. 47; "Arch. gén.," 1846, IV, 12.

Gerhardt: "Würzburger med. Zeitschr.," vol. III.

Rupture of a carcinoma into the left bronchus.

Martius: "Annales de Charité," vol. XII, 1887.

A carcinomatous ulcer began 7 cm. below the bifurcation (?), was 8 cm. long, and broke through into a larger bronchus, which communicated with a cavity in the lower lobe of the right lung.

Freudenhammer: Dissertation, Berlin, 1873.

Esophageal carcinoma perforating the left bronchus.

Jean: "Schmidt's Jahrbücher," vol. CCII, p. 192.

Perforation of an aneurysm of the thoracic aorta into the left bronchus.

Pepper: "Schmidt's Jahrbücher," vol. CCII, p. 185. A similar case.

Damaschino: "Schmidt's Jahrbücher," vol. CCII, p. 186. A similar case.

Peacock: "Trans. Path. Soc.," vol. XXVI. A similar case.

Irvine: "Trans. Path. Soc. of London," vol. XXVIII, p. 63.

Double aneurysm of the aorta, beginning just above the valve and extending to the sixth dorsal vertebra. The left bronchus was compressed by the pressure of the aneurysm, became ulcerated and perforated, but the aneurysm did not rupture. Besides the opening in the bronchus, which was of about the size of a pea, a small abscess had developed. The right lung was emphysematous, the left riddled with small cavities like a sieve.

Moore: "Trans. Path. Soc.," vol. XXXII, p. 33, 1881.

Has a case of ulcer in the right bronchus of inexplicable origin; perforation into the pulmonary aorta and death from hemoptysis.

Kidd: "Trans. Path. Soc.," vol. XXXVI.

Ulcer in the bronchial branch leading to the upper lobe of the left lung; this had broken through into a large branch of the pulmonary artery. Another ulcer in the bronchial wall in connection with a calcified bronchial gland.

Tiedemann: "Deut. Arch. für klin. Med."

Specimen X: Perforation of a traction diverticulum into the left bronchus; specimen IX, into the right bronchus; specimen XVI, into a bronchiectatic cavity of the upper lobe of the left lung.

Ziemssen und Zenker: In "Oesophaguskrankheiten," two cases of traction diverticula, perforating into the right bronchus, pp. 77, 78.

Habershon: "Guy's Hospital Rep.," III, vol. II, 1856.

Case 8: Phthisis. Carcinomatous ulceration of the esophagus, communicating with the left bronchus. Sternal (?) and mesenteric glands swollen. The same author (*loc. cit.*) tells of a remarkable case in which, in an aneurysm of the ascending aorta, an ulcer of the esophagus formed and ruptured into the left bronchus. The relation does not seem clear. The aneurysm did not press directly on the ulcerated place.

Quincke: "Archiv für exp. Path. u. Pharmakol.," vol. XXVIII.

A fistula developed by the degeneration of a caseous lymph-gland, between the left bronchus and the esophagus.

Poland: "Trans. Path. Soc.," vol. XXXVI, 1885. A parallel case.

Causes.—(*a*) *From without inward:*

1. Perforations from inflamed lymph-glands (compare Diseases of the Bronchial Glands).
2. Perforation from new-growths. Perforation of the carcinomatous esophagus.
3. Perforation by an aneurysm.
4. Perforation from abscesses of the lungs, of the mediastinum, of traction diverticula, and of other structures.

(*b*) *From within outward:*

1. Syphilitic ulcers.
2. Foreign bodies (see under these).

Rupture of the carcinomatous esophagus into the bronchi is not so frequently described as we should expect. The condition may develop so gradually, so insidiously, as it were, that the time of rupture cannot be determined. At first there is catarrh, with a peculiar, burning pain deep down in the thorax, coming on immediately after the act of coughing; then we notice that the cough occurs after swallowing; gradually this becomes more and more marked and annoying; bronchopneumonic symptoms, especially in the lower regions of the lungs, make their appearance, and the signs of putrid bronchitis and gangrene of the lungs develop. In a certain number of cases, it is true, the impression made on the patient by the perforation is very violent and sudden. The form in which the expectoration of food with hemoptysis takes place is especially characteristic. Often the signs of pneumothorax also appear. Before we diagnose pneumothorax, however, we must not forget that metallic sounds on the back near the spinal column may also be due to abscess of the lung or to the esophagus itself, if it has aspirated air.

It is interesting that rupture of the carcinomatous esophagus by preference takes place into the trachea immediately above the bifurcation. Among the cases of Petri,* Rebitzer,† and those reported in "Pathological Society Transactions," which I have collected, there are ten perforations into the trachea, five into the left, and one into the right bronchus. The first perforations found in the communication of Vigla, the nature of which is not fully established (however, they are surely not carcinomatous, and probably arise from tuberculous bronchial glands), all three affected the right bronchus (and the trachea).

The duration of life after perforation is short. In Gerhardt's case it was nine days and eleven hours, but I had a case in which life lasted for twenty-two days.

The **diagnosis** of perforation is usually easily established: Expectoration of food, that had been swallowed without coughing, is the pathognomonic symptom. However, we must confess that under complicated conditions things may at times appear very doubtful at the bedside. It may happen that the communication becomes closed and the patient for days appears so well that the physician begins to question the correctness of his diagnosis, especially if he was not called to see the case in the beginning. Under certain circumstances the procedure advised by Gerhardt ‡ may give valuable results. A hollow sound is introduced into the esophagus and the amount of air coming out of it is determined. Very soon the volume becomes so great that there is no room left for doubt.

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* Dissertation, Berlin, 1868. Forty-four cases.

† Dissertation, Munich, 1889. Twenty-one cases.

‡ *Annales de la Charité*, vol. xv, p. 156.

Traction diverticula may rupture into the bronchi in the following way: The diverticulum first breaks into the mediastinum and there forms a decomposing focus, which then breaks into the air-passages. Ziemssen and Zenker describe such a condition in "Oesophaguskrankheiten" in the following classic fashion: "The most frequent occurrence is rupture into one, or perhaps, as the pus cavity generally lies just at the point of bifurcation, into both bronchi. We find, then, on opening the bronchi in the neighborhood of the bifurcation, the walls pierced by one or several, generally pinhead-sized, but also larger openings, from which, on pressure in the neighborhood of the bifurcation, a blackish, putrid fluid exudes into the lumen of the bronchus; by means of this cavity, a fistulous communication is opened between the esophagus and the bronchus, which is not always easily found with the sound on account of the irregular shape of the cavity. Around the opening of the perforation, and extending outward from there, we find the bronchial mucous membrane intensely reddened and loosened, and we have nowhere seen it so finely granulated as in such places (granular bronchitis). The morbid process may be confined to the bronchi without invading the lungs, and it is not to be denied that there is then a possibility of recovery. Perforations of the bronchial wall are often found closed by scar tissue; but these are mostly cases of direct perforation from a softened mass of bronchial glands, without communication with the esophagus, and it is through the latter that the danger is very much increased. The decomposed fluid penetrating into the bronchus may, however, be further aspirated into the lungs, and in the portion of the lung corresponding to the perforated bronchus a fatal pneumonia may develop in consequence of contact with the decomposed fluid. When cavities already exist in both lungs, ruptured diverticulum may establish a fistulous communication with these cavities, etc."

ASTHMA.

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Such different conditions have been at times designated "asthma," and the term is still so frequently used in a careless way, that we cannot introduce it here without an explanation. The fact is that we mean by this term a very characteristic clinical condition which, however, has no definite pathologic basis.

The lack of corresponding pathologic findings is part of the conception of true asthma. Its main symptoms are, however, dyspneic attacks. How easy, therefore, to jumble together a lot of very different things! Nevertheless we are succeeding more and more in dividing them into groups, and we can hope that the confusion in this subject will become less and less, when physicians hold to the well-established fundamental forms and do not class every case of dyspnea which they do not understand as asthma. Numerous pathologic conditions which accompany diseases of the larynx, of the thyroid gland, and of the thymus, and which were formerly regarded as forms of asthma, are now excluded. The same is true of all toxic conditions that simulate asthmatic attacks in uremia, diabetes, leukemia, and Addison's disease. We can also leave out of consideration the hysteric and neurasthenic forms, which spasmodically simulate dyspnea, and we have left only two well-characterized forms to which we now give the name *bronchial asthma*, also called briefly asthma, and *cardiac asthma*, which, through the investigations in the realm of heart diseases, has been classed with stenocardia and nervous palpitation among the neuroses of the heart.

The definition of asthma is very difficult, especially as I desire to avoid entering into the hypothetic assumptions of authors concerning the cause of the disease. The definition given by Jürgensen might best answer the purpose, as it simply mentions the main symptoms of the disease: "We characterize as bronchial asthma spasmodically occurring conditions of dyspnea, in which expiration is especially labored, the number of respirations is more frequently increased than diminished, sibilant and hissing râles of the fine vesicular type become audible, and which are attended by the production of emphysema." Still, I believe that we ought not to overlook the characteristic sputum. The significance of this has been so positively established by the works of Leyden and Curschmann, that Fraenkel * is justified in enumerating suddenly occurring hyperemia of the blood-vessels, swelling of the mucous membrane of the finer bronchioles, and the production of a specific secretion as the essential symptoms of the disease.

The syndrome of Brissaud: "Spasm, dyspnea, vasomotor disturbance," will be more pleasing to many. The last causes the peculiar secretion. [Thus far theories only exist to explain the nature of this affection. The trend of opinion seems to be that with the neurotic element, a *bronchiolitis fibrinosa* or *bronchiolitis exudativa* (Curschmann) exists.—Ed.]. But I should like to see distention of the lungs added to these three phenomena. If we wish to say anything in our definition concerning the nature of the disease, which would certainly be justifiable, since a mere enumeration of symptoms can never really characterize a disease, we might say: *By asthma we understand a neurosis in the distribution of the respiratory nerves, which leads to attacks of dyspnea, with a peculiar secretion and distention of the lungs.*

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* Eulenburg's "Real-Encyklopädie," 1885.

Etiology.—In the consideration of the causes of asthma, we distinguish the fundamental cause from the predisposing and determining factors. The fundamental cause is not known. According to our present views, it is perhaps a pathologic change of the respiratory center, and therefore to be sought in the medulla oblongata. The predisposing causes are both hereditary and acquired, and hence we see that the ranks of asthmatics are especially recruited from families in which gout, obesity, epilepsy, and migraine are prevalent, and that several asthmatics are found in nearly every large family. The male sex is certainly more predisposed than the female. (Is this due to the influence of gout?) The members of certain professions, lawyers, professors, and clergymen, are said to be especially susceptible. The poor are said to suffer from it less frequently than the rich. Of predisposing diseases, which may be acquired accidentally, we have chiefly those which affect the respiratory tract: nasal, laryngeal, tracheal, and bronchial diseases. But these also differ greatly in importance. For the present our chief attention will be given to the nasal affections. Incidentally, I would call especial attention to the great danger involved in the infectious diseases which attack these regions, at the head of which is whooping-cough, next measles, and then influenza.

Of statements about cutaneous or eczematous asthma also there is no lack, the significance of which Sée reduces as drastically as appropriately to its proper dimensions.

To mention all possible weakening factors, from neurasthenia to cold, would be a useless multiplication of words.

The determining causes were first sought in the climatic conditions. Asthma appears to be especially frequent in England, the south of France, and North America. For certain patients, certain places are especially dangerous. Van Helmont suffered from an attack every time that he went to Brussels. I knew a patient who was free from the disease in Berlin, but was attacked in Schöneberg. Winter is more favorable than summer, but there are patients who suffer most in the winter; windy days are feared more than calm weather. We have the case of a man who always had an attack during the new moon, and another who was always attacked on Monday; quite peculiar and quite personal factors, idiosyncrasies, are of importance. The same thing is to be noticed regarding certain kinds of dust, the odor of fresh hay, lamp soot, powder of ipecac [feathers.—

Ed.], and even the smell of horses [or cats.—Ed.]; the fragrance of the rose, of the violet, and of the mignonette may cause asthmatic attacks. Trousseau himself had an attack when oats were being measured in his presence. Besides the dust of the oats, he ascribes the occurrence of the attack partly to a simultaneous psychic irritation, which existed in this case (a suspicion of larceny was involved); moreover, we have to consider that Trousseau had formerly been asthmatic. Among such individual predisposing causes we will mention the following: Dust of hemp (odor in the shop of a rope-maker?); dust from maize straw; linseed meal; scammonium powder; Trousseau gives a large number of such idiosyncrasies among the determining causes. Bresgen describes a very carefully observed case of hay-asthma, which would certainly indicate that the grasses are only determining factors, while the predisposing factor is a nasal mucous membrane affected with catarrhal inflammation and therefore easily irritated.

Those cases have excited especial interest in which the attack of asthma could be caused reflexly by some stimulation of a neighboring mucous membrane, especially that of the nose, so that even touching a certain place might bring on an attack. We are reminded by this of the odors above mentioned. But more distant mucous membranes may also be considered in this connection: that of the stomach (*asthma dyspepticum*), that of the ear, that of the intestine (*asthma verminosum*), and of the uterus (*asthma uterinum*). *Asthma gravidarum*, of which very different cases have been described, may be especially considered here. Thus, Salter tells of a case which began with conception and lasted in monthly attacks until confinement in the eighth month. Another patient had asthma as a girl from her sixteenth year, but it disappeared on her marriage and occurred again during each pregnancy. In a third case the asthma became permanent in the patient's thirty-seventh year, while it had formerly occurred only at the time of confinement. In Croom's case asthma occurred with pregnancy in a woman who had previously been healthy; the attacks increased rapidly and finally became so violent that they produced an abortion. After this, the asthma disappeared again.

Finally, there is a large number of cases in which we do not generally succeed in showing any cause. These cases have been designated "essential asthma," while the former are known as cases of "symptomatic asthma." In reality, however, the two are identical; in the one case we recognize the cause, which, acting on the periphery, affects the diseased center and brings on the attack; in the other case we do not recognize any such exciting cause.

There are certain points of view that are therapeutically important, in those cases in which symptomatic asthma can be diagnosed.

The question may arise whether there are injuries, which, affecting the center directly, may bring on the asthmatic attack. From the nature of things, this question cannot be unconditionally answered either in the negative or in the affirmative. That the cases which are mentioned as examples permit of many objections there is no doubt.

Fright and anger are often charged with the attack, when no other cause can be discovered. Psychic trauma has already become a doctrine. It is as popular an etiology in nervous diseases as is taking cold in catarrhs, and is always given when words are needed and knowledge is lacking. A trauma can always be found nowadays.

[A. Fraenkel's* reports on the causes of asthma are most interesting and have led to much discussion. With Curschmann, he seems to establish both a neurotic and an inflammatory element in the etiology. He first discusses the bronchial muscle-spasm theory as a cause of asthma. He agrees with Biermer that practically all the signs and symptoms, including the characteristic form of dyspnea, the expanding of the lungs, and the low position of the diaphragm, can be explained by this theory. However, it cannot be asserted from the experiments on animals that resistance to respiration can be caused by bronchial spasm, sufficient to account it the sole cause of an asthmatic attack in man. This question could only be answered if there existed a purely spastic form of bronchial asthma. Fraenkel has never seen such a case, and says there is always some change in the bronchial mucous membrane. From his previous work and from the autopsies on two cases of bronchial asthma he concludes that there is always an epithelial inflammation of the mucous membrane, and that the process originates in the bronchioles, in which a remarkably abundant epithelial desquamation takes place. This latter is sufficient in itself to account for the dyspneic attacks; and the bronchial changes alone can account for all the signs and symptoms of the asthmatic attack, except that it is impossible, in consideration of the fact that the process in the bronchioles takes a certain time for its completion, to lay to its account the suddenness of the appearance of an attack, or even the paroxysmal exacerbation of a continuous dyspnea. And so the attention is turned to the nervous system. Weber makes the nervous system responsible in that he says that the process in the bronchial mucous membrane is a vasomotor turgescence, and Fraenkel finds this hard to refute. From the fact that asthmatic attacks can follow emotional excitement, or the irritation of single peripheral nerve regions, as the olfactory nerve and the nervus trigeminus, or reflexly from irritation of distant nerve regions, as from the uterus, he concludes that the nervous system must play an important rôle in the pathogenesis of the disease. As a rule, the catarrh, the bronchospasm, and the influence of the nervous system work hand in hand. So asthma is really a neurosis; indeed, a reflex neurosis. This assumes, of course, an abnormal condition of the nervous system, either inherited or acquired, which either limits itself to an increased irritability of the entire mucous membrane surface of the respiratory tract or of certain circumscribed diseased spots, or is a part of a general neurasthenia.—Ed.]

As the course of the disease is so different and the etiology so manifold, a **classification** has been undertaken in order to facilitate the arrangement and grouping of the observations. Asthma is termed, according to the region from which the exciting cause emanates, nasal, pharyngo-laryngeal, bronchial; to which are added the dyspeptic, the sexual, and the uterine forms of asthma. These names will always be used in order to indicate briefly a pathologic condition and its probable point of origin. This classification for the present can have value only in so far as, by the establishment of an etiologic factor, it strengthens us in our prognosis and therapeusis; but the same is true in all diseases. Numerous cases always remain, in which we are led to consider a form as idiopathic or neurasthenic asthma, since the origin of the reflex irritation remains unknown.

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* "Die Deutsche Klinik am Eingang des 20ten Jahrhunderts," 4 Lief., 1901.

Brügelmann distinguishes a form which he names intoxication-asthma. According to this author, this form is due to the fact that there is some obstruction to the respiration, the spasm being brought on by carbonic acid intoxication. It is obvious that this form can be very easily included among the nasal, pharyngeal, and bronchial forms, and that the distinction is often quite artificial.

Course.—The disease rarely begins at first in its classic form. There is a preliminary stage in which the patients have other troubles, which, however, correspond in that they all tend to bring on bronchitic symptoms. Such patients very readily get bronchitis. But otherwise the symptoms of this stage are those that are peculiar to the individual diathesis. Nervous symptoms, tendency to eruptions of the skin are mentioned, but a comprehensive picture of this stage has not yet been presented.

Gradually, however, the classic asthmatic condition develops: the characteristic attack occurs at certain intervals. Many patients have a certain premonitory sensation, a kind of aura, which warns them of the approach of an attack; thus, for example, a sensation of pressure and constriction behind the sternum, a coated tongue, a peculiar taste, and slight headache. However, the patient lies down and goes to sleep, but two or three hours later he is roused by a sudden want of breath. His face becomes bloated and cyanotic. There is a sense of heavy pressure on the chest, which seems to suffocate him; he rises from his bed, opens the window, in order to have fresh air, draws it in with all the strength of his respiratory muscles, but gets no relief. He sits down, he crouches down, he thrusts his face into his hands and his elbows on his knees. His face becomes red, tears and saliva flow, the perspiration runs from his forehead, one can hear him breathe, there is a loud hissing and whistling sound with every breath; the respirations are themselves surprisingly long, and it is expiration that is most labored. The chest appears expanded, and we can easily convince ourselves that the edges of the lungs are drawn further downward, that the heart is displaced, and the liver dulness pushed down; evidently the whole diaphragm is depressed and its excursions are small, especially in proportion to the efforts which the patient makes. The auxiliary muscles of respiration also, the scaleni, sternocleidomastoid, and trapezius muscles, stand out prominently and do not rest even during expiration. The shoulders are drawn up and the elbows or arms extended in order to make use of the pectorals and the serrati during inspiration. The recti are powerfully contracted at each expiration, but they move little, and their effectiveness is extremely limited. On auscultation, numerous dry râles are heard, most distinctly during expiration. In this pitiable condition the patient remains for one or many hours, until gradually relief comes; he begins to cough and expectorates mucus, at first very little and with great difficulty; the mucus is extraordinarily tenacious, but it becomes more and more abundant and frothy, and at length the patient is greatly relieved. Now a considerable quantity of urine is often passed, from which a large amount of urates are precipitated—and the attack is over. The patient falls into a refreshing sleep and the next morning feels quite well. Only a certain weariness, an indolent expression of countenance, a certain bloated appearance of the face, are still to be noticed; in many cases the patient feels perfectly well.

Often, however, the attacks recur with astonishing punctuality, at the same hour, at the same minute, for several, yes, for many days, one after the other, reminding one of a masked intermittent fever, until finally the attacks become less frequent and there is a longer period of well-being between the paroxysms. Such groups of attacks may often be repeated at longer or shorter intervals, until the disease gradually passes into the terminal stage. Chronic conditions then develop, especially emphysema; the breathing is slightly but permanently labored; typical attacks, however, become more and more rare, and finally lose their original type entirely—the patient has by gradual degrees become emphysematous, with occasional exacerbations; these exacerbations bear only a faint resemblance to true asthmatic attacks; they are just as distressing, but they have no longer the same typical course; beginning, duration, and ending are quite indefinite. Gradually the heart becomes involved and the condition develops which is described as the last stage of emphysema.

From this typical course the disease shows many **variations,** which more or less modify or even completely mask it. Often the attacks are not nocturnal, but may occur in the daytime. In the symptomatic form which occurs after a definite injury (hay-asthma) the attacks are, of course, diurnal. The duration of the attack also varies; although this was placed at two or three hours in the description of the typical case, yet there are milder cases, as well as cases in which the attacks drag along for days and even weeks. And, strange to say, there are cases in which the attacks are repeated in more and more rapid succession, so that they might be compared with the status epilepticus.

In a certain number of cases it happens that inspiration is much more labored and attended with much more exertion than expiration, or that both appear equally laborious. In still other cases, there is no marked spasm of the inspiratory muscles, and yet the respiration is labored, and hissing râles are heard over the whole chest. A spasm of the bronchial muscle must be considered as an important factor in the causation of these cases.

In many cases that remarkable condition develops which is described as ictus laryngeus. The patients lose consciousness, fall, have some epileptiform movements, which, however, only last a moment; immediately they rise again and act as if nothing at all had happened. There is no feeling of weariness or dull weakness as after epileptic attacks. The only danger that threatens the patient is that he may hurt himself in falling.

Under certain circumstances it has been observed that the attack is accompanied by spasms of other muscles; so there may be a spasm of the glottis, or even a true epileptic attack.

The catarrh also is very different in different cases. It may be very abundant from the beginning, and the condition is then described as *asthma humidum;* it may continue during the intervals between attacks. The catarrh may also so predominate that it alone represents the entire attack. The patients suddenly have an attack of expectoration, which lasts for some hours without any dyspnea, to disappear and reappear at regular intervals, after the manner of asthmatic attacks.

In many cases the attack begins with a severe coryza; the patients sneeze constantly before the real attack of asthma begins. But it may not go beyond the stage of coryza, and we then no longer have bronchial, but nasal asthma. This leads us to the consideration of the abortive forms. A patient of Trousseau, who had formerly suffered from asthma, began to have only regularly recurring attacks of sneezing and lacrimation. In other cases, this famous clinician asserts, he has correctly predicted that the patient was about to have an attack of asthma from the characteristic attack of sneezing, coryza, and coughing.

As masked forms of asthma, we may mention febrile attacks, which come on suddenly with bronchitis and disappear in a few hours; as they occur especially in children, they are easily misinterpreted, and we only discover later that we had to do with the beginning of asthma. Another masked form consists in attacks of apprehension, which, however, may as well be classed with cardiac asthma.

Trousseau has given especial attention to the asthma of children, and we find in his "Clinique médicale" two very fine examples described. I myself have also observed a case, which at first appeared to me to be a case of whooping-cough with remarkably sudden and violent onset, until, made suspicious by the too brilliant results of my treatment, I came to the correct diagnosis.

I will here mention the classic case of Trousseau, because I know of nothing more instructive: Father healthy, mother hysterical. One of the children is attacked quite suddenly by symptoms which suggest bronchopneumonia. Over the chest are heard numerous crepitant râles, and there is extraordinary shortness of breath. Flying blisters are applied to the chest and recovery ensues in three days. Three months later there is another attack; datura stramonium is burned in the room, and on the next day the child is well.

In old people, as well as in children, the asthma is generally masked and shows the so-called catarrhal type. We find a chronic bronchitis with occasional exacerbations, with arteriosclerosis, changes in the myocardium, and emphysema; and it is often a purely arbitrary matter whether we shall ascribe an asthmatic character to the affection or not. If the use of potassium iodid is beneficial, we are accustomed to consider that fact as a sign of the existence of an asthmatic condition as the fundamental cause of the attacks.

As a form of symptomatic asthma, which has often excited great interest, I will mention so-called **hay-asthma.** This is very infrequently met in Germany and France, but is much more wide-spread in England, and still more so in America.* It consists of asthmatic attacks, which are very similar to those already described, except that the odor of hay, and especially the irritation of the air-passages caused by the pollen of certain grasses at the time of blooming, is regarded as the determining cause. The recognition of this factor was certainly a considerable advance, but it appears somewhat narrow to ascribe the attacks to the pollen alone. For, while at first the pollen of only certain definite kinds of grass was believed to be causative, it soon had to be recognized that hay-asthma occurred in places where this grass did not grow, so that very different kinds of grass had to be admitted as possible causes. Finally cases enough were found, which had certainly begun as hay-asthma, but which later occurred at any time of year and in the most different places, without the presence of any hay. We have thus the most varied transitional forms from the true hay-asthma to our ordinary cases. We can, therefore, consider the pollen of the grass as a determining cause for the production of the attack, but cannot regard it as the essential factor, as has been done.

The disease was first described by Bostock † under the name "Sommerkatarrh," and Blakeley has studied carefully the pollen of the grasses, rye, barley, oats, wheat, alopecurus, poa, and finds them especially instrumental in bringing on the attacks. [See also Morrill Wyman's monograph and Beard's studies.—Ed.]

The investigations of Phoebus established the fact that in England it is generally the hay (*Anthoxanthum odoratum*), in Germany generally the blossom of the rye, that brings on the attacks. In America, *Ambrosia artemisiæfolia*, which does not occur at all in Europe, but grows very extensively in the United States and blooms in August and September, is especially feared.

Sée is also of the opinion that in hay-fever we have an asthmatic affection in which the pollen certainly acts as a determining factor, but not necessarily the only one. He knew a patient with hay-asthma who, after having suffered during the summer for twenty years, finally developed permanent dilatation of the right heart, and two of whose three daughters suffered from classic asthma, the attacks coming on both in summer and in winter. Healthy people are as little liable to become asthmatic from the pollen of grass as healthy apothecaries are from the powder of ipecacuanha.

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* The best collection of histories of the disease in this form we owe to Phoebus, "Der typische Frühsommerkatarrh," Giessen, 1862.

† We find the older literature in Hirsch's "Handbuch der historisch-geographischen Pathologie," or in Phoebus.

We usually distinguish two groups of hay-fever patients: The *catarrhal*, who suffer from coryza, conjunctivitis, bronchitis, supraorbital neuralgia, and occasionally from true febrile movements; and the *asthmatic*, who have typical attacks with expiratory dyspnea, sibilant râles, and distention of the lungs. But there are many transitional forms. Hay-asthma is distinguished from other forms by the fact that the symptoms of irritation and even inflammation of the nasal mucous membrane are so constant; sneezing, feeling of tickling and prickling, coryza, which in the beginning of the attack is dry, but is soon accompanied by an abundant secretion, epistaxis, dulling of the sense of smell, and swollen appearance of the nose itself. Further characteristic features are that the attack occurs at a certain definite time (about the first hot spell, end of May or beginning of June), that the asthmatic attack lasts about one to three months and then disappears and leaves the patient for the rest of the year entirely free. In many of these patients a second attack occurs in the latter part of the summer. After that, however, recovery is complete, any disturbance of it being due to accidental complications. In many cases the attacks continue the even tenor of their way no matter where the patients may stay, but in the majority of cases the attacks cease, and begin again when the patients return to a true hay-fever region. But this is also true in other forms of asthma.

Closely allied to hay-asthma are those forms which are induced reflexly by irritation of the nasal mucous membrane, and which have claimed so much attention that we now never treat an asthma patient without first examining his nose. Hay-asthma is certainly in many cases a nasal asthma. Different specialists in the diseases of the nose have reported brilliant examples of the curing of the asthma by operation on the nose (Daly, Mackenzie, Roe, Hack). In truth we not infrequently find in asthmatic patients polyps and hypertrophy of the mucous membranes, especially in certain places, as in the anterior portions of the lower turbinate. This latter pathologic condition, still more than polyp, predisposes to asthmatic attacks, which are in every respect like those described above. In the tissue of the polyp, and in the hypertrophied mucosa, great numbers of eosinophile cells have been found.

Among the numerous cases of nasal asthma, we find also single cases in which it is stated with great definiteness that the attack has its seat on only one side of the chest. Sommerbrodt describes two cases, in which the attacks had their seat on the left side only, and one in which the attack, while it had formerly occurred equally on both sides, once, according to the patient's statement, attacked only the left side. Unfortunately these cases were not subjected to physical examination.

After the nose has been treated the patients are wonderfully relieved, and the attacks may remain absent for a long time. But the recovery is by no means permanent; the asthma returns whether the nasal affection does or not. If the nasal affection recurs, the second treatment does not usually have the brilliant results of the first. There are also cases in which the first operation is of no advantage,* a fact which will not surprise us much.

It is quite natural that areas, the irritation of which has a reflex influence on the bronchial system and on the respiratory muscles in general, should be found elsewhere than in the nose, and that we should have a nasopharyngeal, a pharyngeal, a tonsillar, and a laryngeal asthma. Here it is generally not a polyp, but only a circumscribed swelling or granulation of the mucous membrane that is regarded as the starting-point of the asthma.† A characteristic case of uterine asthma is also found in Brügelmann's collection.

The younger the individual, the greater is his chance of perfect recovery. The attacks become more infrequent, atypical, and finally disappear. In older people, especially in those beyond the forty-fifth year, recovery is the exception. The rule is rather a change to emphysema, chronic bronchitis, and cardiac asthma. If certain determining causes are known and can be avoided, recovery is often easy and certain.

Asthma may begin at any age; cases in the first year of life even are not at all rare, as Salter in his statistics reports not less than 11, and 60 in the first decade! It is certainly doubtful how reliable the diagnosis was.

In general, asthma that does not develop until after the fortieth year has a special tendency to pass into the third stage. This is shown by the fact that the patient no longer feels perfectly well between the attacks, but always remains somewhat short of breath and suffers from chronic bronchitis. All the signs of emphysema develop. But we must not forget that an emphysematous character is often found in asthmatic patients, in whom a true emphysema has not yet developed, for the elasticity of the lung is still retained, and in the intervals between the attacks we can easily recognize the perfect rise and fall of the diaphragm. This soon changes and soon a certain weakness appears; the heart becomes dilated and a tendency to swelling of the lower extremities becomes noticeable. Expectoration also assumes quite a different character; it is no longer characteristic, the spirals are wholly lacking or are to be found but rarely; the sputum often becomes entirely mucopurulent or exclusively purulent. In many cases we now find the sputum which we have described as characteristic of fibrinous bronchitis: long, dendritic coagula, which represent casts of the bronchial tubes. In many patients bronchiectasis, which is peculiar to emphysema of the lungs, develops to an extraordinary degree.

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* Two observations of Gentilhomme cited by Joal. Fraenkel has one case among a large number of successful ones (not described in detail).

† Brügelmann gives several important case-histories.

In no small number of patients bronchial asthma gradually passes over into *cardiac asthma;* the enlargement of the heart to the right is complicated by tachycardia and a surprisingly impure, dull, weak first sound; the liver becomes enlarged and the gastric function suffers; dyspnea is now very severe at times, but it is not an expiratory or inspiratory dyspnea, merely an excessively rapid and superficial respiration.

Symptoms.—(*a*) *Of the Attack:*

The Sputum.—This is quite characteristic in the true asthmatic attack. The rule is that expectoration comes at the end of the attack; it is only exceptionally that it precedes the attack, or occurs in the interval between the attacks. In the third stage, expectoration may become chronic, but even at this time it may be lacking for longer periods of time. True asthmatic sputum is grayish-white, never yellow, and formed of thick, tenacious mucus, capped by a fine foam. It may be quite abundant, 100 c.c. and more, but there are cases with very scanty expectoration, and occasionally moist and dry asthma have been distinguished. Long ago, before the microscope was used in studying the sputum, tough, fibrillar masses had been observed, which were regarded as casts of the bronchi (Lefèvre); but besides these larger ones, there are also finer plugs, which can be better recognized with a pocket lens; when compressed between two glass slides, they are found to be elastic. With a somewhat higher magnification, the peculiar spiral arrangement of these plugs is recognized. With a magnification of more than 200 diameters, we find cells, and often crystals, which are known as Leyden's crystals. The spirals consist usually of a central thread, which is solid (although it may show vacuoles in certain places), around which the mucous masses are

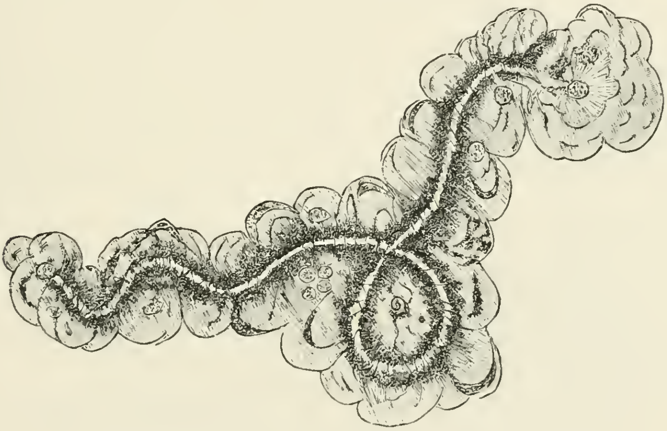

FIG. 8.—Perfectly formed spiral, with distinct central thread.

arranged in a very graceful, spiral fashion. The pictures vary greatly. At times the central thread is very perfectly formed; at other times it is indistinct and only suggested or quite unrecognizable. The spirals around it may be isolated and irregular, but the most beautiful spiral bundles in the most regular arrangement may also be seen. The central thread may also appear naked (without the spiral) and very perfectly developed. It is itself then disposed in a spiral. At its extremity, it occasionally runs into very fine threads and also sends out slender lateral processes into the surrounding mass. According to Schmidt's description, it can no longer be doubted that it consists of mucus. The central thread is therefore not essentially different from the surrounding spiral; it is that part which first forms in the finest bronchi and attains relatively the greatest firmness. This fact is

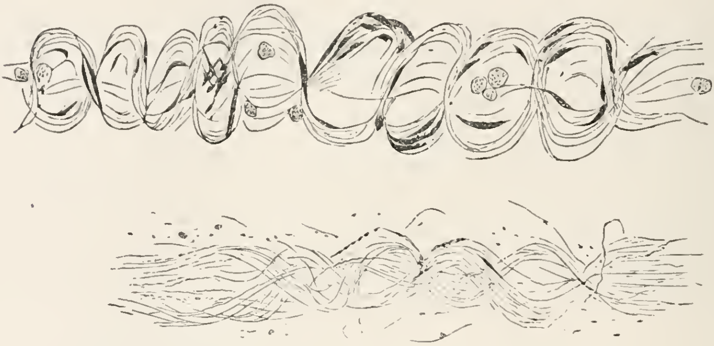

FIG. 9.—Rudimentary forms of spirals without central threads.

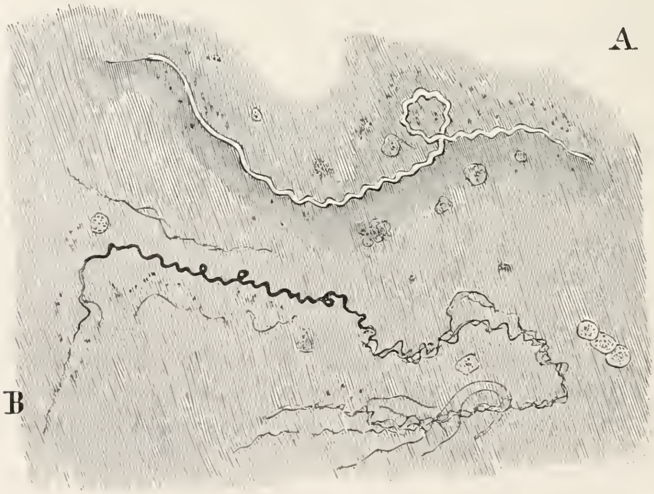

FIG. 10.—A, Free central thread, perfectly developed; B, rudimentary central thread.

indicated by the so-called transitional forms of Schmidt. Hans Ruge has gone still further in the art of staining the spirals. He asserts, and emphasizes the assertion, that they consist of mucus, and that the central thread is a greatly thickened and twisted thread of mucus.

The investigation of a very favorable case by Schmidt showed that the epithelium of the smaller bronchi was well preserved and that the lumen was filled with a mass of mucus. The winding of this mass could be very nicely followed; it was found to be smallest in the finest bronchioles, and became thicker and thicker, as the larger bronchi were approached. In the medium-sized bronchi, where cartilage plates begin to be found, there were seen in a cross-section several such winding figures, formed by the union of several smaller bronchi. As typical central threads are found in small bronchi, where there are no glands, the threads cannot be regarded as a secretion of the bronchial glands.

The spirals which contain air are an interesting form; they are tubes with a mere suggestion of a spiral arrangement, or not even a suggestion, which end in true spirals containing no air.* If the air-containing portions alternate with those which do not contain air, very peculiar figures result. Occasionally such an air-bubble can, by pressing on the cover-glass, be made to move to and fro in the interior of the spiral; but the movements are slight, and we are not justified in assuming that there is a preformed canal within the spiral.

The production of such remarkable forms as these spirals necessitates a special secretion of the finest bronchi and a revolving force acting upon it. The peculiarity of the secretion consists in its tenacity and, chemically, in its tendency to deposit asthma crystals. The catarrh, which produces this secretion, is correctly spoken of as a special form, and Curschmann has given it the name *bronchiolitis exsudativa,* as he himself says, in order to have a neutral designation. I must confess that to me this designation appears almost too neutral. I share Curschmann's view perfectly, that we have here an exudative process which can be considered neither as a simple catarrh nor as a fibrinous bronchitis. I even go so far as to consider the process quite specific, and without hesitation use the designation "asthma-catarrh."

Attention has recently been called by ophthalmologists to structures found in different types of keratitis, which are quite similar to these spirals. This similarity has even been called striking.† So far as I can gather from the aforesaid literature, these are fibers of mucus, which are rolled up by the motion of the lids on the concave surface of the cornea, and thus gain an appearance that approaches that of the spirals. I do not venture to decide whether there is not a considerable difference. For instance, the ophthalmologists describe and sketch in their forms homogeneous, brightly granular deposits which are not known to me in the asthma spirals. On the other hand, no ophthalmologist mentions asthma crystals or eosinophile cells. I therefore believe provisionally that the similarity between the asthma spirals and the keratitis threads is entirely superficial. In both cases, however, a tough mass of mucus acquires by a rolling movement a peculiar appearance, which is characteristic, and so the comparison of the two forms may be interesting.

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* Curschmann has described these. I myself have never observed any of this form.

† Leber, "Fädchenkeratitis," XIV. Versammlung der ophthalmologischen Gesellschaft zu Heidelberg, 1882. Uhthoff, *Gräfe's Archiv,* Bd. XXIX, 3, S. 181. Fischer, *Gräfe's Archiv,* Bd. XXXV, 3, S. 202, 1889. Czermak, *Zehender's klin. Monatsblatt,* Bd. XXIX, S. 56, 1891.

A great deal of ingenuity has been used in explaining the spiral winding. All authors agree that the peculiar tenacity of the sputum is a fundamental condition of their production, for even if a revolving force should produce such crystals in another medium, they would not hold together, but would always fall apart. But the most different ideas have been expressed in regard to what this revolving force is. [It is suggested by Osler that the currents of air produced by the ciliated epithelium may be rotary. This movement, with the combined action of the spasm of the bronchial muscles, may give a spiral form to the mucus.—Ed.] I am forced to assume that the terminal branches of the whole bronchial tree end in spirals. For when we consider that at each inspiration the lungs are distended, and that the bronchi must follow this distention, can it be imagined that long, straight tubes should be distended longitudinally? It is much more probable that they resemble corkscrews, and it is thus readily explained how the bronchi can follow the distention of the lungs. A very beautiful metallic cast of the bronchial tree, which I saw at the Leipzig Pathologic Institute, shows this corkscrew-like course of the larger branches of the bronchi and suggests the assumption that the finest branches have a similar course. If, now, a very tenacious mass is crowded through a spiral tube of this kind, it must necessarily assume a spiral shape. The anatomic conditions and the tenacity of the mucus thus cause the formation of the spirals. It is now understood also how the fibrinous casts which originate in the fine bronchi occasionally show a spiral arrangement.

If one has by chance at his disposal a very tough sputum, he can easily convince himself that by revolving a thread of mucus a characteristic spiral with beautiful central thread can be made, as Sänger, Gerlach, and Lenhartz have stated, and as I have often seen myself. We cannot, of course, match the perfect beauty of nature's handiwork, but the mechanism becomes quite clear, even to the formation of the central thread, which cannot be regarded as anything essentially different; it is only the most strongly twisted portion of the mucofibrillar mass. Fibrinous coagula may also occur in asthmatic sputum, as we could unquestionably show by the proper use of the Weigert and triacid stains.

[Fraenkel * recently investigated the subject and concludes that Curschmann's spirals are composed almost entirely of mucus, as has been discovered by the use of a modification of the Biondi-Heidenhain

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* "Die Deutsche Klinik am Eingang des 20ten Jahrhunderts," 4 Lief., 1801.

stain. The spirals are formed in the smaller bronchioles—of a lumen of 0.15 to 0.03 mm. The ciliated cylindric cells of the epithelial exudate become considerably increased in size, and a few of these are very much longer than the corresponding elements of the normal mucous membrane exudate. These elongated cells secrete tenacious mucus, which remains in contact with the cells; three or four of these become partially free, and lie together in the lumen of the bronchiole. They become spindle-shaped, and a lash-like process from the end toward the bronchial wall is inserted between the normal cells. This process is all caused by the production of a large amount of very tenacious mucus, which clings to the cell for a long time, and by the to-and-fro current of air caused by respiration. The stream of air so acts on these cells that they are drawn out of their positions and made into the described elongated threads. Gradually in this way, and by a rotation on the long axis, from the mucus and the cell-thread the Curschmann spirals are formed.—Ed.]

In the mucus of the spiral fiber are found cells, among which eosinophile cells can be demonstrated in large numbers by the employment of appropriate stains. [The origin of the eosinophiles has been the subject of considerable inquiry, as well as their diagnostic significance in sputum. Schmidt's * attention was called to the fact that not only the sputum, but also the nasal secretion, which was profuse after an asthmatic attack, contained very many eosinophilic leucocytes. He then investigated the mucous membrane of the nose, and found it infiltrated with innumerable leucocytes, a great number of which contained eosinophile granules. The mucous membrane in cases other than those of bronchial asthma rarely contained any eosinophiles. Von Leyden (*l. c.*), though he agrees with the other investigators in regard to the increase of eosinophiles in the blood as well as in the sputum of asthma cases, suggests that their origin in those cases may be a local one in the nasal and bronchial mucous membrane, and not in the spleen and bone-marrow, where these cells are thought to be usually formed. He also points out the intimate relation between the occurrence of Charcot-Leyden crystals and eosinophiles, and attributes it to the fact that a lymph exudate takes place into the alveoli, and that in a peculiar way the formation of eosinophilic cells and Charcot-Leyden crystals is here brought about. A. Fraenkel † believes that the eosinophiles, which are mostly mononuclear, come from the blood, as similar cells were observed in the small vessels in the neighborhood of the bronchi. Such cells were also seen to be apparently making their way between the epithelial cells of the bronchi. While Mandybeer (*l. c.*) grants the probable origin of some of the esoinophiles in the glands of the bronchial and nasal mucous membrane, he believes, with Neusser, that some of them are derived from blood, as there is an increase in eosinophiles in the blood during and after an attack. According to Neusser (quoted by Mandybeer, *l. c.*) the sympathetic system governs the output of eosinophiles, and their increase is brought about by irritation of the peripheral nerve-ends in the bronchial mucous membrane, causing an irritation in the end apparatus in the bone-marrow and the other places of origin of the eosinophiles. All agree that eosinophilic sputum is not pathognomonic of bronchial asthma, but, as von Leyden says, on account of the frequent and constant association of eosinophilic sputum with bronchial asthma its presence has some intimate relation with this form of disease.—Ed.] Schmidt was able to show mast-cells also in the sputum of asthmatic patients. But they were always present in small numbers.

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* "Zur Kenntniss des Asthma bronchiale," *Centralblatt für klin. Med.*, No. 25, 1891, p. 473.

† *Deutsche med. Wochenschrift*, April 26, 1900.

In a certain relation to the eosinophile cells is the occurrence of crystals, which were long ago discovered by Leyden and the nature of which is still doubtful. They have hitherto been identified with spermin crystals, but Cohn brings convincing proof against this view. They are very distinctly characterized by their rhombic form, but they are not so very easily recognized, as they are not strongly refractive. The cross-section is hexagonal, as first shown by Koch and verified by Cohn. Leyden finds that there is a very close relation between the occurrence of the crystals and the occurrence of eosinophile cells; he even believes that the crystals originate at the cost of the Ehrlich cells.* Von Noorden observed that these crystals increase greatly in number after exposure to the air.

The crystals are found in irregular groups, which are distinguished by their greenish-yellow color and greater density from other flocculent admixtures in the transparent ground-substance. These groups are not infrequently massed together into regular plugs, which may even form thread-like rolls. They are with difficulty pressed out under the cover-glass into a dry, friable mass with a faint luster, and appear as a closely crowded layer of brownish, granular, decomposed cells, between which the crystals in question are scattered in greater or less abundance. The cells themselves show in their interior partly a myelin-like degeneration, and partly larger or smaller masses of black, granular pigment; they remind us of alveolar epithelium, but have a peculiar yellowish-brown faint luster. As the spirals are the favorite place for the crystals to congregate, the former may show even macroscopically a yellow or even yellowish-green color. If the crystals are found massed together here and there in the spiral, then the latter appear grayish-white with yellow spots. The plugs and masses described may undoubtedly be regarded as rudimentary spirals. After a diligent search, we may find distinct transitional forms, as described by Curschmann. The crystals themselves are perfectly colorless, easily broken, and therefore fall to pieces on pressure. They may vary much in size; we cannot generally find them with a low magnification, but should use a power that magnifies at least 180 to 200 times. Even then the search may be in vain, and at last we may succeed in getting a positive result with an immersion lens. The

* *Deutsche med. Wochenschr.*, 1891, p. 1085.

crystals are usually somewhat larger when the attack has lasted for some time, say two or three days; then we also find crystals which are in a defective condition. By keeping the asthma sputum in a moist chamber, we may occasionally succeed in separating out crystals where there were none before. So there must be a substance in the sputum which is not always expectorated in the form of crystals. We find a similar behavior on the part of tyrosin in purulent sputum. Our crystals certainly do not consist of tyrosin. As long as they were considered identical with spermin crystals, they could be regarded as crystals of the so-called Schreiner's base; but as we are now convinced that they appear quite different, we shall have to reject this view also. Until further investigation, therefore, we must declare the chemical nature of these crystals to be unknown.

[Lewy takes up the question whether the Charcot-Leyden crystals are identical with the Böttcher spermin crystals or not. In an historical note he traces the investigations in regard to the two crystals and details the various opinions, which up to 1891 agreed upon their identity. The author found that the spermin crystals in general have not sharp angles, but that they are slightly convex or S-shaped; and that these crystals may appear as octahedrons, double pyramids, or prisms. The Charcot-Leyden crystals are rhomboidal, or in the form of double pyramids,—which heretofore have been regarded as octahedrons,—and hardly ever are found as prisms. Charcot-Leyden crystals are usually quite small, about 20 to 30 μ, and exceptionally as long as 100 μ; the spermin crystals are decidedly larger, often as long as 900 μ and more, and are visible to the naked eye as fine shining needles. After considerable study of the crystal form, the writer concludes that the spermin crystals are monoclinic prisms or a combination of monoclinic prisms and pyramids; while the Charcot-Leyden crystals are hexagonal double-pyramids, double cones, or spindles. In regard to their behavior in polarized light, the Charcot-Leyden crystals are found to be optically uniaxial, the Böttcher crystals biaxial. There are also slight differences in their chemical reactions, and Lewy considers the difference in their behavior toward an iodin-potassium-iodid solution an important aid in the establishment of the non-identity of these crystals. The almost constant coincidence of eosinophilic cells with Charcot-Leyden crystals, and the fact that these cells are rarely found with spermin crystals, have some bearing on the argument that these crystals have nothing to do with each other. Lewy concludes from all of these facts that the Charcot-Leyden crystals are not identical with spermin crystals.—Ed.]

The importance of Leyden's crystals has been many times attacked and belittled, but careful investigations show that they are found practically constantly in asthmatic sputum. Leyden himself found them six times in seven cases, and the last case could not be thoroughly investigated. Curschmann failed to find them only four times in 38 cases. Ungar investigated 23 cases, all with positive results; Schleussner investigated 9 cases, of which 8 were positive. In looking for these crystals it must always be borne in mind: (1) That they are very small, so that we must even look for them with the immersion lens; (2) that the crystals are often not found in fresh sputum, while they are easily found in sputum that is partially dried (hence they separate out on standing).

Besides these crystals, Ungar found also calcium oxalate. As to cells, Noorden has several times, although not constantly, found some containing yellow and brown pigment, which on the addition of hydrochloric acid and potassium ferrocyanid became blue (hemosiderin cells). Part of these pigment cells show the eosinophile granules, but the great mass of the eosinophile cells present in the sputum were free from pigment; another part contained neutrophile granules, and were therefore declared to be altered white blood-cells; while the third part, about the half of all, showed no granulation, and were regarded as epithelial cells. Usually the pigment cells stood in no relation to the spirals, but occasionally they took an important part in the formation of the spirals.* Traces of blood are not rare in the sputum of asthmatic patients, and therefore the occurrence of hemosiderin cells can easily be explained.

Many times the mucous secretion is not limited to the bronchial mucous membrane, the mucous membrane of the larynx and pharynx and the mucous and lacrimal glands secreting more abundantly.

The classic *time of the attack* is just before midnight (as in gout), yet it is occasionally observed at other times (examples are given by Trousseau). During the subsequent course the attacks may, of course, become more infrequent, or they may increase up to several attacks in a day.

The time of year also at which the paroxysms occur is in many cases remarkably definite. True hay-asthma is most remarkable in this respect, occurring every year at the beginning of the summer heat (the end of May, or the beginning of June in Germany).

Attitude.—The patients rarely remain quietly in bed; Bamberger's patient did so †; they often sit with their heads bowed and supported on their hands, or they can endure the attack only in the standing position. However, they always avoid the more violent movements, as if they knew how much the production of carbonic acid was increased by such movements.

Respiration.—The respiration is dyspneic, but the number of respirations is not increased; on the contrary, it is often diminished on account of the marked lengthening of the expiration. Inspiration is relatively short, but deep and strong; the upper part of the thorax is vigorously raised, often more vigorously than in normal breathing, while the lower portion of the thorax arches forward but little and, in the highest grade of the asthmatic attack, sinks back somewhat toward the spinal column.‡ At the same time, the upward

* Excellent illustrations are found in Noorden, *l. c.*

† *Würzburger med. Zeitschr.*, vol. VI, Nos. 1 and 2.

‡ Riegel in Ziemssen's "Handbuch," vol. IV, p. 328. On the other hand, in

movement of the thorax is increased. The inspiratory muscles contract vigorously. Bamberger says that inspiration shows nothing different from ordinary, strong, somewhat labored inspiration. But it is very ineffectual; in his own case the increase in the girth of the thorax, with the tape under the axillary space, was only 1.5 to 2.5 cm., while at the xiphoid process it was only 1 to 1.5 cm. Expiration lasts two, three, or four times as long as inspiration; all the auxiliary expiratory muscles are convulsively contracted, the abdominal muscles so much so that they appear like boards, and the urine and feces may be expressed involuntarily by these convulsive muscular contractions. No respiratory pause can be observed, the distinctly visible expiratory movement beginning as soon as the inspiratory movement ceases.

Tracheoscopy shows a reddening of the trachea.

Percussion.—Signs of distention of the lungs, downward displacement of the liver, indistinctness of the heart dulness, downward displacement of the heart, slight displacement of the borders of the lungs during inspiration and expiration, change in the percussion note (*Schachtelton*, "box-tone," Biermer*). These conditions gradually return to the normal after the attack. The displacement of the lungs can be well demonstrated after the paroxysm, if there are no complications.

Auscultation.—Vesicular breathing is lacking, being weakened or concealed by râles. The respiration is accompanied by peculiar sibilant noises, which become especially intense during the greatly prolonged expiration. Toward the end of the paroxysm the râles usually become moist.

Cough does not belong to the attack, and is to be observed only occasionally with the predominance of catarrhal conditions. More frequently it happens that it joins itself to the catarrhal secretion which accompanies the end of the attack.

Coryza is especially observed in the nasal form of asthma, and either introduces the paroxysm, terminates it, or it may simply take the place of asthma; in other words, it may represent a masked asthma. With violent sneezing, the mucous membrane suddenly becomes swollen and reddened, and a large amount of serous, fluid transudate that does not form threads is discharged. Fraenkel saw three teacupfuls discharged in a short time. The whole course is represented as a vasomotor neurosis.

Fever is not usually a part of the asthmatic attack; however, there are cases in which the temperature suddenly rises to a considerable height with the attack; this is observed especially in children. Such forms of febrile asthma usually pass in time into the simple form of asthma. In hay-asthma febrile disturbances appear to be frequent,

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Bamberger's case measurements of the periphery of the thorax give the result that during the attack the circumference at the level of the ensiform cartilage is greater, and that at the level of the axilla is less, than between paroxysms.

* *Sammlung klin. Vorträge*, No. 12, 1870. This term is, as a rule, used so arbitrarily, and the impression is so entirely a subjective one, that I reject it for the present.

as may be assumed from the name "hay-fever," and Phoebus mentions all kinds of febrile symptoms (chill, chilly feeling, dry skin, febrile sweat, feverish, considerably accelerated pulse). But I cannot find in the literature any authentic observation of the temperature. As a rule, on the other hand, a surprisingly low temperature is observed during the attack. The peripheral portions of the body generally appear cool and to a greater or less degree cyanotic.

The *action of the heart* is accelerated during the attack, the pulse being often 120 or more.

In Bamberger's patients the action of the heart was strengthened and the impulse was felt over a greater area. The pulse-rate was lessened (from 92 to 60). During the paroxysm the pulse becomes smaller, and often disappears entirely at the height of the attack.

In general, we find the *pulse* remarkably small during the attack; this is explained by the obstruction to the circulation in the lungs, which undoubtedly accompanies the hindrance to the respiration.

Consciousness may in rare cases be lost during the paroxysm. When this is the case, indications of opisthotonic and tetanic convulsions are sometimes observed. Such cases form transitional stages to true epilepsy, the relation of which to asthma is shown in their common hereditary character. Ictus laryngeus has already been mentioned.

Headache and severe *neuralgias* in different regions of the trigeminus, especially of the supraorbital nerve, are mentioned in all forms, but especially in so-called hay-asthma. In this connection, dizziness and roaring in the ears are also mentioned. Rheumatic pains may be present in various regions of the body.

Urine.—During the attack large quantities of urates are frequently precipitated from the urine.

Skin.—At times there is severe itching and a wide-spread eruption of urticaria (Phoebus, p. 31). It is also stated that the attack may be replaced by an urticarial eruption (p. 57).

The *features* are bloated, especially the nose in hay-asthma; this member, as well as other portions of the face, frequently itches severely.

(*b*) *In the intervals between the attacks:*

Bronchitis. A feeling of pressure and constriction in the chest. All kinds of nervous symptoms.

Under this head the development of emphysema in the later stages of asthma must be considered. Sooner or later the heart regularly suffers in true asthmatics; the cardiac changes go hand in hand with the development of emphysema; the right heart becomes dilated.

The *digestive organs* also participate in the disturbance; we find especially a tendency to flatulence and constipation; dilatation of the stomach is also mentioned. It is explained by a weakened condition of the diaphragm, which in the intervals between attacks does not offer sufficient resistance to the diminution in size of the thoracic cavity and to the lengthening of the abdominal cavity. (If this were correct, the high position of the diaphragm must continue between the attacks.) The expiratory muscles also lose their normal power between the attacks, and this favors the stagnation of the fecal masses.

In the *blood* of asthmatics the eosinophile cells have been found to be increased. Noorden even established a relation between the severity of the attack and the increase of eosinophile cells in the blood during the attack. There is no doubt that there is also an increase of these cells during the intervals between the attacks. [Ewing states that Gabritschewsky found in 3 cases, 11 to 22 % of eosins, Fisk 14.6% in one case, and Billings 53.6% out of 8300 cells.—Ed.]

After long continuance of the disease, asthmatics acquire a *characteristic appearance;* the strongly arched chest, with which is contrasted the marked emaciation, the raised shoulders, between which the head hangs slightly forward, with staring, slightly bulging eyes, present a peculiar picture. All older asthmatics are cachectic and anemic.

Complications.—Noorden observed a complication with a peculiar psoriatic *eruption of the skin.* It has also been shown that many asthmatics have suffered in their youth from eczema; this indicates one of the predisposing factors which is called by the French the dartrous diathesis. Sée had among 28 asthmatics with skin eruptions 10 children who had eczema of the head; 15 in middle life with eczema, lichen, and psoriasis; and 3 old people with eczema or prurigo.

The complication with *bronchitis* may occur in a peculiar way, not, as usual, forming a symptom of the asthma, but in such a manner as to represent a true complication. Under the influence of this complication the asthma itself may entirely cease during the continuance of the bronchitis, or it may become extremely mild, as in the example described by Trousseau.

A certain number of cases are complicated with *tuberculosis;* either the asthma is added to the tuberculosis or the latter is added to the asthma. A certain reciprocal exclusion has been assumed to exist between the two diseases, and this is also true. When the two diseases combine, each gives up a part of its peculiarities: the asthma, its characteristic paroxysmal character—the attacks become weak and indistinct and pass over into indefinite dyspneic conditions; the tuberculosis, its progressive character—it is prolonged and degenerates into fibrous phthisis. The association of asthma and tuberculosis is certainly very rare. Brügelmann says quite positively that as long as any one has asthma, he cannot become tuberculous. He relates the interesting case of a tuberculous woman who suddenly began to have attacks of asthma; but they were pseudoasthmatic, for they disappeared the instant the patient expectorated a gland that had undergone cheesy degeneration. He also describes an instructive case of pseudotuberculosis in an asthmatic (p. 35).

The complications with *gout* and with *urticaria* ought to be especially mentioned. These diseases occur with the asthma in the sense that they may take its place or coexist with it. Urticaria usually alternates with the asthma, and this alternation is so marked that asthma has been regarded as an urticaria of the bronchi. Such a case is mentioned by Trousseau,* but unfortunately the description is too summary. Gout, on the other hand, usually replaces the asthma; it appears with advancing age, after the asthma has disappeared and appears to be cured. Yet attacks of gout and asthma may alternate. *Migraine* generally acts in a similar way. Trousseau † describes the interesting case of a man who was first asthmatic, then contracted gout, and finally suffered from migraine. Graves gives the case of a small boy who suffered from asthma and then had an attack of gout, since which time he has remained well. But gout and asthma occur by no means frequently in the same patient. Lecorché has investigated the histories of 150 gouty patients, and found that 5 of them were asthmatic; Sée investigated 370 asthmatics and found 14 who also had gout.

The relation to *epilepsy* may also be mentioned here. Sée states that he has seen 7 such cases in which one disease had no influence on the other.

Attacks of *neuralgia* of the trigeminus and of the sciatic; epileptic, hypochondriac, and maniacal attacks have also occurred in place of asthma.

Complications with heart lesions and with phthisis are found in Fraenkel (*l. c.*, p. 2235); complications with skin eruptions, psoriasis, and eczema, as mentioned by Thorowgood (*l. c.*, p. 94).

Nature of the Disease.—Authors are now agreed [see Fraenkel's report, p. 229.—Ed.] that asthma is a neurosis, that it cannot be explained by bronchitis, by the peculiar form of catarrh, or by the peculiar constituents of the secretion. As in all diseases occurring in paroxysms,—epilepsy, migraine, spasm of the glottis, stenocardia,—the nervous element predominates. We physicians, according to an easily understood hypothesis, find this nervous element in a center. In what way the latter is affected pathologically is not explained. We can only assume that injuries acting upon it leave behind some impression which does not disappear, but remains latent for a certain time; there is an accumulation of injuries, and when this has reached a certain magnitude, some apparently unimportant, peculiar factor causes an unburdening or explosion, and this is the paroxysm. These injuries are generally conveyed by the paths which act most directly on the respiratory center, the sensory nerves of the respiratory tract. They may, however, although much more slowly, be introduced in all possible ways. The opportunity for the accumulation of the injuries is given by a certain predisposition of the nervous system, as in epilepsy or migraine, which may be congenital or acquired (even cultivated!). The explosion manifests itself in a convulsion of numerous muscles; authorities still disagree as to whether it is to be ascribed to a spasm of the bronchial muscles or to a spasm of the inspiratory muscles, especially the diaphragm. I believe that both are right, and that often both systems are attacked, and sometimes one and sometimes the other. But vasomotor fibers are also affected by the reflex. The secretion which occurs almost regularly at the close of the attack is regarded as the consequence of reflexes acting on secretory or vasomotor fibers, but the mechanism is not explained. Moreover, secretion of the bronchi has not as yet been demonstrated after stimulation of certain fibers, although undoubtedly the same rule holds good for the bronchial glands as for other glands. [Aufrecht's experiments * show that the unstriped muscle of the finer bronchi is made up of a thick layer of circular fibers, and a much weaker layer of longitudinal fibers. These two layers are intimately interwoven. In asthmatic cases in which the bronchial mucous membrane is intact, which are to be explained by reflex spasm of the muscle-fibers, the contraction of the strong circular layer overcomes that of the interwoven weaker longitudinal layer, which under normal conditions prevents a narrowing of the lumen of the bronchi. Thus contraction of the bronchioles is brought about. In asthmatic cases which follow upon a catarrhal condition of the finer bronchi and bronchioles, the weaker longitudinal layer of unstriped muscle-fibers suffers more by the inflammatory process than the circular layer, which, again, constricts the lumen. In these latter cases the condition is one of increased circular contraction, due to interference with the action of the longitudinal antagonists, rather than to a muscular spasm, which occurs in the first class of cases.—Ed.]

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* *Loc. cit.*, p. 488.

† "Clinique," éd. 4, p. 486.

That the nerves of the blood-vessels take part in the asthmatic attack cannot be doubted. Schmidtborn concludes that there is spasm of the muscular layer of the arteries of the lungs, and tries to deduce the whole symptom-complex of asthma from this factor.

The attack is brought on in a purely reflex way by some determining cause. The connections of the diseased center with the higher psychic centers have an important influence; for the latter, on the one hand, possess the power to counteract the attack, while, on the other hand, they may favor or even provoke it. Brügelmann rightly emphasizes this influence of the will; if the will-power is strong, reaction and its consequences are proportionately delayed; on the other hand, the feebler the will-power, the sooner must reaction take place. The disease is possible in its most marked form only when the will-power is greatly altered, especially weakened, as in neurasthenia.

With all this knowledge, we are still far from understanding the true genesis of this remarkable convulsion, which so powerfully attacks the inspiratory muscles.

Diagnosis.—Dyspneic attacks apparently occurring suddenly always demand a careful differential diagnosis. If positive proof of the occurrence of a paroxysm with the characteristic secretion is insisted upon, the correct diagnosis will in most cases be made. If the physician is called in at the time of the attack, he is very apt to make mistakes. The great importance of noting the expiratory dyspnea, especially insisted upon by Riegel, should be once more emphasized. By this means attacks of spasm of the glottis, edema of the glottis, paralysis of the extensors of the glottis, tracheal stenosis, and central causes (especially tumors of the medulla oblongata and of the upper portion of the spinal cord, a point that has not received sufficient attention in the case-histories at our disposal) can be excluded with certainty. Expiratory dyspnea occurs, outside of the disease under discussion, in bronchitis, in the presence of foreign bodies and bronchial stenosis in general, in emphysema, and in cardiac asthma. The possibility of spasm of the diaphragm should also be considered, especially in hysteria. The distinction between bronchitis and asthma presents peculiar difficulties in children, as infantile asthma is very atypical.

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* *Deutsches Archiv für klin. Med.*, vol. LXVII, Nos. 5 and 6.

Enlargement of the bronchial glands must always be thought of, especially in children. In these cases a short, dry, irritative cough usually exists, with signs of dulness between the shoulder-blades; as the children are generally tuberculous, we should expect exacerbations of fever, night-sweats, and emaciation.

As to foreign bodies, we must always consider that we can often find the right track, by inquiring into all the details of the anamnesis. Yet we have seen cases enough in which the foreign body has reached the bronchi without the knowledge of the patient or of those immediately around him. For other forms of bronchostenosis it will always be possible to find a cause, and, especially, to demonstrate the participation of a lung.

As asthma and emphysema often imperceptibly merge one into the other, the differential diagnosis must rest on the demonstration of the characteristic sputum. Another decisive factor is obtained most simply by means of the spirometer; for in emphysema the capacity of the lung is considerably reduced, while in the asthmatic patient, in the interval between the attacks, the capacity must be nearly or quite normal. When emphysema complicates asthma, the spirometer must decide as to the emphysema, and the microscope as to the asthma.

Cardiac asthma is, according to Fraenkel,* a disease depending on a diminution of the functional power of the left ventricle, which manifests itself by sudden attacks of dyspnea, cyanosis, and accelerated heart action. It favors the occurrence of edema of the lungs, which is absent in bronchial asthma. The dyspnea is equally strong in both phases of respiration; the diaphragm is not depressed; the heart symptoms must be distinctly evident. The characteristic sputum of asthma is absent and eosins are not found in the blood in the same high percentage as in asthma.

In aortic aneurysm there are asthmatic attacks from pressure on the vagi; these should be considered in this connection. I have not been able to find an exact description of them: In these attacks the differential diagnosis from cardiac asthma assumes special importance.

Hysterical spasm of the diaphragm may simulate asthma. It is characterized by a short inspiratory movement, with retraction of the yielding structures, as in laryngeal stenosis. Then follows a quick and short expiratory effort, accompanied by a sobbing sound. True dyspnea is not at all a feature of this form.

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* *Charité Annalen*, vol. v, 1880.

Observations from the more recent epidemics have shown that even the onset of influenza may present symptoms very similar to those of an asthmatic attack, so that the two could not be distinguished by ordinary methods of investigation alone, without a longer time for observation. Microscopic examination of the sputum, and eventually the examination for bacilli, would, however, clear up the diagnosis.

The diagnostic importance of the eosinophile cells cannot be too highly esteemed; when they occur in large numbers, they are always a considerable indication in favor of asthma, especially if affections of the heart and kidney can be excluded. For the differential diagnosis from cardiac asthma they are, however, of no assistance at all. The diagnostic value of the asthma crystals is the greatest of all, although they may also occur in fibrinous bronchitis. The diagnostic significance of spirals is on a level with that of the crystals, although they have likewise been demonstrated in simple bronchitis, in fibrinous bronchitis, and in pneumonia. [Osler's experience and that of others is quite contradictory of the above. The first author states he has never seen true spirals in pneumonia or in bronchitis. Gollasch * has found oxyphilic leucocytes in the sputum of acute and chronic bronchitis, and also in the nasal secretions occurring during the course of those diseases. In tubercular sputum and that of bronchitis fœtida he did not discover any eosinophiles. Von Leyden † found a single eosinophile in the sputum of a consumptive, though the sputum of the other phthisical cases examined exhibited no eosinophilic cells. He discovered a few eosinophiles in the scanty sputum of a case of croupous pneumonia with delayed resolution, and also in the nasal secretions during an attack of acute coryza. Schmidt ‡ states that oxyphilic cells are found in the fibrin clumps in the sputum of fibrinous bronchitis, as well as in the diphtheritic membrane. He has not found any such leucocytes in the sputum of phthisis pulmonalis or of suppurative bronchitis. Mandybeer § and Gabritschewsky ‖ cite cases of emphysema, in the sputum of which eosinophilic leucocytes were almost constantly found. Mandybeer (*l. c.*) has also found them in influenza sputum. He agrees with the other investigators that the sputum of tubercular and pneumonic cases rarely, if ever, shows any eosinophilic cells.—Ed.]

Conditions resembling an asthmatic attack may occur in a great variety of diseases, especially if the diagnosis is hastily made and every attack is declared offhand to be asthma, after the manner of the laity.

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* "Zur Kenntniss des asthmatischen Sputums," *Fortschritte der Medicin*, Bd. VII, 1889, p. 361.

† "Ueber eosinophile Zellen aus dem Sputum von Asthma bronchiale," *Deutsche med. Wochenschrift*, No. 38, 1891, p. 1085.

‡ "Beiträge zur Kenntniss des Sputums, insbesondere, des asthmatischen, und zur Pathologie des Asthma bronchiale," *Zeitschrift für klin. Med.*, Bd. XX, 1892, p. 492.

§ "Vorkommen und diagnostische Bedeutung der oxyphilen und basophilen Leukocyten im Sputum," *Wiener med. Wochenschrift*, No. 7, 1892, p. 256

‖ *Archiv für experimentelle Pathologie und Pharmakologie*, Bd. XXVII, 1 und 2 Heft.

I should not dwell on this point, if it were not so self-evident as to belong in every manual, that cases of so-called lead-, alcohol-, malarial, or syphilitic asthma in reality have nothing whatever to do with asthma. I do not, of course, pretend to deny that a syphilitic or an alcoholic subject may have asthma. If a reliable history of such a case were presented to me, I should declare it a coincidence of two diseases.

Examples are also mentioned of hysteric asthma, in which I cannot find any similarity to true asthma. The same is true of uremic asthma. On this principle diabetic, leukemic, and Addison's asthma might be described.

Treatment.—The treatment of this disease has been extremely variable. By experimenting with numberless drugs, some fairly good, and a few excellent results have been obtained empirically; but the results gained by a deeper knowledge of the disease are far greater. The treatment, so far as is possible, should be directed toward removal of the cause, and with such treatment brilliant results may often be obtained. The determining causes are especially amenable; if they are often trivial or even ridiculous, their removal can nevertheless be brought about under certain circumstances. People who have an asthmatic attack whenever they have to do with powdered ipecac or with hay can certainly avoid these irritating causes. Better results are secured by attending to the predisposing causes, and searching for asthma-points, especially in the nose, but also elsewhere in the body, and by removing their pathologic irritability and their pathologic exciting influence; here, then, the rhinologist especially, but also the laryngologist, the gynecologist, and the genito-urinary surgeon, may accomplish much.

The treatment of *nasal asthma* is considerably simplified by the use of cocain. [Such treatment must be employed most cautiously.—Ed.] The mucous membrane is desensitized and so-called asthma-points—that is, points on the mucous membrane which on pressure with the sound cause definite reflex expressions—are searched for. If such points are found in polypi, in swellings of the mucous membrane or erectile tissue, or in other alterations, they are immediately treated locally, and, if necessary, by an operation. But the results of the direct nasal treatment are often very slight, or, while apparently successful for a time, they are of very short duration. Then we can say with certainty that it is not a case of true nasal asthma; that the nasal involvement is only incidental or does not exist at all, and we must therefore look for other causes. Brügelmann found that always when he had cauterized true asthma-points, a marked febrile reaction set in in the evening, which, in very sensitive patients, might continue for several days.

It is certain that we must think of treating the nose, but it is equally certain that we must not expect too much from nasal treatment, and that asthmatics may suffer, and have already suffered, much harm at the hands of too energetic nasal therapeutists. Much mischief can be done with the galvanocautery. [With this the editor cordially agrees.] The patients do not complain to the specialists who have treated them, and these, therefore, unwarned, report the most brilliant results. One must be very careful in his judgment of the literature.

Among the predisposing causes is a pathologic excitability of the imagination, with a corresponding weakness of the will-power. The thought: You will have an attack if you do this or that, or if this or that happens, makes the patient a victim to the most ridiculous determining causes. In these cases psychic treatment is necessary; the physician must assert his authority, and even hypnosis is not to be shunned. Numerous examples cited by Brügelmann show the great value of psychotherapy, which in severe cases is indispensable.

Of course, it would be best, if we could, to attack the fundamental cause of the disease, but there is no prospect of discovering it within a reasonable time. Something can, however, be accomplished in this respect also. In the consideration of the nature of the disease it was found that an injury may affect the respiratory center at some point in the central nervous system; in such a way the center receives certain impressions and utilizes them improperly, so that the injury gradually accumulates, until a kind of unloading or explosion takes place. This is in many respects similar to epilepsy, migraine, and hysteric convulsions. It should be treated on the same principles, according to which we in such cases train and spare the central nervous system. Gymnastics of the respiratory center, exercising and regulating the act of breathing, and the general invigorating measures represented by the cold-water treatment, are both to be especially considered as auxiliary means.

That *psychotherapy* and the treatment of the central nervous system in general can often be used with great advantage under the careful supervision of the physician in private institutions is sufficiently evident to show that such forms of treatment may in many cases be urgently recommended to the patient.

It is of all things important to limit the number of attacks as soon as possible, and thus give the nerve-center time for recuperation. This may often be secured by a change of residence and a diligent search for and removal of the predisposing causes. It is, however, undoubtedly aided by attending to the determining factors concerning which the individual experiences of the patient must decide, and by the use of medicines and similar auxiliary measures.

Among *internal remedies* potassium iodid should be considered; in large doses, given internally, it often brings surprising relief, although I do not believe that a patient will recover by the use of this medicine alone. Next to potassium iodid, but much inferior in value, is arsenic. [Patients in whom attacks occur when debilitated or just below par have been relieved by the use of ascending doses of nux vomica. In the editor's experience this remedy in properly selected cases is as valuable as arsenic or the iodid of potassium.—Ed.] Then follows the main army of less useful drugs, most of which act on the imagination alone, and which I only mention in order to explain their usefulness in producing a psychic impression: tincture of lobelia, quinin, strychnin, hyoscin, flowers of zinc, silver, bismuth, iron, potassium bromid, alkaline phosphites, castoreum, camphor, asafetida, and flowers of sulphur [*Euphorbia pilulifera;* Tull].

Inhalations are of value when there is bronchitis, since the patient is greatly relieved if the bronchitis is checked. Simple warm water vapor or carbonic acid water, Ems water, and other alkaline waters are often very agreeable to the patient. Breathing with Waldenburg's apparatus is a good method to compel the patient to perform respiratory gymnastics and to let his respiratory muscles act vigorously, as we know they are forced to do. This is, then, an essentially different method from the inspiration of compressed air in the pneumatic cabinet, which is highly praised by Lazarus; the latter combats the tendency to emphysema, and is really an admirable means of checking the catarrh.

Specific drugs have also been inhaled. Thus, Sée praises pyridin in glowing terms. It is a colorless, very volatile fluid; 4 to 5 gr. (about one dram) are to be poured into a saucer which is placed in the middle of a small room, and the patient is to breathe this medicated air for twenty to thirty minutes three times daily. As the great name of the originator of this procedure has not caused it to be further recommended, we may consider it worthless; and as it causes dizziness and nausea, we must pronounce it harmful.

Inhalations of turpentine and carbolic acid have often been recommended, even before Sée suggested pyridin. Their value is, however, not to be compared to that of the inspiration of the vapor of warm water and moist steam. To carry out these various inhalation methods a suitable mask may be made to order by any instrument-maker. Schäffer especially recommends the *faradic current;* and this is confirmed by Fraenkel and Bresgen. Schäffer believes that the starting-point of the reflex stimulation is always somewhere in the respiratory tract, and according to the place where he assumes it to be, he places the electrodes on both sides of the neck, under the lower jaw, 2 cm. in front of the angle of the jaw, or at the level of the thyroid cartilage in front of the sternocleidomastoid. The current should not be too weak and should be used twice a day for one-quarter to one-half hour.

The climate is entirely a matter of individual experience. Thorowgood considers that of London especially beneficial, because there is so much carbonaceous matter in the atmosphere! Some prefer moist, others a dry atmosphere; one fears the sea, and another the mountains. Brügelmann finds the climate of Paderborn extremely favorable. [For young subjects, southern California or Florida is useful. Older persons with favorable vascular conditions (aged twenty-five to thirty-five years) improve in Colorado. Hay-fever subjects derive relief from residence during the summer in New Hampshire, Maine, or the Adirondacks.—Ed.] It must not be forgotten also that in early times certain sulphur baths were popular among asthmatics, as: Baths at Vienna, Weilbach, Harrogate, Amélie-les-Bains (very favorable climatic conditions), Cauterets, and Raillère. At the present day we hardly ascribe any special value to them, but empirically the climatic conditions have certainly been found favorable. Even when actual climatic treatment is out of the question for external reasons, something can be accomplished by insisting on having plenty of pure, fresh air by day, and especially in the bedroom at night. The fear of taking cold is responsible for an incredible amount of injury, especially among the lower classes.

The physician must not forget to speak of the *diet;* many severe cases have been cured by a strict regimen. Vegetarianism, it seems to me, has found many enthusiastic advocates in England. Pridham recommends the following diet: In the morning, breakfast at 8 o'clock; ½ pint of tea or coffee and 60 grams (2 ounces) of stale bread. At noon, about one o'clock, 60 grams of beef or mutton, 60 grams of stale bread or boiled rice. In the afternoon, at 4 o'clock, ½ pint of brandy and water, or sherry and water, or toast and water, according to preference. In the evening, at 7 o'clock, 60 grams of meat and 60 grams of bread.

Such a denutrition cure can, of course, only be used with caution and in selected cases; especially for corpulent people who live well and have good nerves, as ladies frequently have who imagine themselves to be very nervous.

Milk cures have also proved truly efficacious in many cases.

The effort to shorten and relieve the patient's attack is generally most successful with the help of morphin injection and chloral hydrate. The two medicines are equally recommended, and it will have to be decided according to the accompanying circumstances whether, in a given case, the one or the other is the more advisable. But in older cases it is found that the patients either abuse these remedies or that they are entirely opposed to them. Many other suggestions have therefore been made. In the first place, inhalations of amyl nitrite are warmly recommended, but there are not many who can boast of permanent or brilliant results from this drug. Belladonna, the subcutaneous injection of atropin, cannabis indica, chloroform and ether inhalations, and potassium bromid in large doses may be considered. The inhalation of turpentine has also been recommended, but it is of little value during the attack.

The smoking of cigarettes, prepared with stramonium, has been greatly lauded. Trousseau gives the following prescription: leaves of belladonna, 0.36 (6 grains); hyoscyamus, 0.18 (3 grains); phellandrium aquaticum, 0.06 (1 grain); and stramonium, 0.18 (3 grains), carefully stripped from the ribs, dried, and treated with a trace of extract of opium. The cigarette papers are then filled with the mixture, the papers being especially prepared by washing in cherry-laurel water. The most valuable constituent of these cigarettes is unquestionably the small amount of opium. Yet the cigarettes made of camphor and stramonium also enjoy considerable repute. The smoke must be really inhaled; the mere blowing away of the smoke will suffice only in the mildest cases. Many patients get relief by smelling ammonia from a flask. (Painting of the throat with ammonia is objectionable.) Trousseau tells of a ship captain who noticed no trace of an attack when his ship was loaded with guano. The inhalation of the smoke of so-called niter paper should really be included under the head of ammonia inhalations; carbonic acid, carbonic oxid, and cyanogen are at the same time evolved, and potassium carbonate and potassium nitrate may also be carried into the air-passages mechanically. But even during the attacks I believe the vapor of warm water is far ahead of all kinds of smoke and vegetable dusts. A simple remedy that should always be tried consists in the immediate use of hot hand- and foot-baths, which often afford considerable relief in a very short time. The efficacy of these methods is, so to speak, individual; and many people experiment on themselves with the most wonderful remedies, among which may be mentioned brilliant illumination with a number of lamps or a brisk trot on horseback. How strange is the story told by Graves of the two asthmatic patients whom he visited in one day! the first ascribed his attack to the smoking of his chimney, while the second let his chimney smoke and felt better for it.

Emetics boast a number of very ardent admirers: Lebert and Seitz use them in cases in which the attack is prolonged. Thus one might even be tempted to try ipecacuanha, tartarus stibiatus, or apomorphin. For those who demand a prescription, I will mention one given by one of our greatest clinicians, the above-mentioned Graves: Tr. hyoscyam., 2.0 (30 minims); Acet. scillit., 2.0 (30 minims); Vin. ipecacuanha, 2.0 (30 minims); Camphora, 0.2 (3 grains); Vin. 10 (2.5 fluidrams); Aq., 20 (5 fluidrams), to be taken in one dose. The camphor will, of course, be almost entirely precipitated.

[**The Use of Heroin in Asthma.**—Strube [*] reports two cases of bronchial asthma, in which mild attacks were relieved. The rate of respiration and coughing are lessened, and sleep is often induced. Tauszk [†] observed one case in which severe dyspnea was relieved, and the attacks did not return. Leo [‡] used heroin in a number of cases of bronchial asthma with success, and in one case he reports a probable cure. Medea [§] had rather poor results with heroin. Rosin [||] had no better results than those obtained with codein or morphin, and encountered unpleasant after-effects, such as vertigo, nausea, and severe headache. Bloch [**] had only good results, and concludes that heroin has a sedative action upon the respiratory tract. North [††] reports marked relief in asthma, following the use of heroin. Floret [‡‡] has naught but praise for heroin in the treatment of this disease. Harnack* has exaggerated the toxicity of heroin, as is shown by the collective experience of the many observers who have used it safely. Manges† says that heroin, and especially the hydrochlorid, is superior to any drug now being used in the treatment of bronchial asthma. The paroxysms are shortened and the interval between attacks is lengthened. He used the hydrochlorid subcutaneously in $\frac{1}{12}$ to $\frac{1}{6}$ grain doses, repeated in an hour if necessary. Then heroin is administered by mouth every four to six hours to insure sleep. Cardiac and renal asthma are benefited by the drug, but morphin is of more service here. The consensus of opinion seems to speak for the efficacy of the drug, while recommending that, as a derivative of morphin, it should be dispensed with discrimination.—Ed.]

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[*] *Berliner klin. Wochenschrift*, No. 45, 1898.

[†] *Orvoci Hetilap*, 1898, No. 80.

[‡] *Deutsche med. Wochenschrift*, March 32, 1899.

[§] Morgagni, June, 1899.

[||] *Therapie der Gegenwart*, June, 1899.

[**] "Die Heilkunde," 1899, Heft 8.

[††] *Amer. Med. Compend*, June, 1899.

[‡‡] *Therapeutische Monatshefte*, June, 1899.

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* *Münch. med. Wochenschrift*, 1899, Nos. 27–31.

† *New York Med. Jour.*, Jan. 20, 1900.

EMPHYSEMA

AND

ATELECTASIS.

BY

FRIEDRICH A. HOFFMANN, M.D.

EMPHYSEMA AND ATELECTASIS.

PULMONARY EMPHYSEMA.

The term emphysema has long been applied to certain morbid conditions in which the lung appears excessively distended by an abnormal volume of air. Our present conception of the word derives from Laennec, who made a sharp distinction between two varieties: interstitial and vesicular pulmonary emphysema. Vesicular pulmonary emphysema is produced when the air, without deviating from the normal air-passages, distends first the alveoli, which are the least resistant structures, and later the infundibula, and even the bronchi. It is this vesicular emphysema that will be discussed now. Under ordinary circumstances the elasticity of the normal lung is adequate to the varying degrees of distention to which the organ is subjected, and it maintains its integrity until extreme old age to the extent of being able to satisfy the demands of life. But if, as the result of disease, the distention becomes excessive and unusually abrupt, or if the walls of the alveoli suffer some pathologic change that interferes with their nutrition, or if, finally, these two events coincide, then the lung fails to regain its normal volume and remains permanently distended. If the condition is only temporary and there is a possibility of recovery, it is called distention of the lungs, or alveolar ectasia, or acute emphysema, and represents a comparatively insignificant process. It may, however, under certain circumstances be the forerunner of the condition in which the lung fails to return to its normal volume and a permanent morbid state, with a tendency to progressive development, takes place. This is genuine chronic emphysema, and is the condition usually meant when the term pulmonary emphysema is used without qualification.

If a large portion or even the entire substance of both lungs is involved in this form of emphysema, and the basal disease sinks into the background, the clinical picture of so-called **primary emphysema** is produced. In reality this primary emphysema is as much a secondary emphysema as all other forms of the disease, and it is admitted by all authorities that the most common cause is found in chronic bronchial catarrh. A true primary emphysema exists only in theory. This question will be referred to again in the section on etiology.

The essential features of what is called primary emphysema are that it practically involves the entire substance of both lungs, although the degree of intensity varies in different areas; that it dominates all other pathologic phenomena when once it has become fully developed; and that it may be the starting-point of various other severe morbid processes. It is owing to the second peculiarity that the disease is regarded as an independent and well-defined entity, and the term primary emphysema is justifiable. The tendency to involve the entire pulmonary area would justify the term **diffuse emphysema,** which is also sometimes used in contradistinction to partial emphysema.

In addition to primary emphysema, there are other forms accompanying severe alterations in the lungs, which at once reveal themselves as essential morbid conditions and in which the emphysema is accordingly recognized and described as secondary from the outset. These forms are, as a rule, partial, so that **secondary** and **partial emphysema** are practically used indifferently, although, as has already been said, primary emphysema is also a secondary condition. It has therefore become a matter of habit to distinguish between primary emphysema and secondary or partial emphysema; but the most popular term is **compensatory emphysema,** the idea being that the contraction and injury of portions of the lung brought about by phthisis, bronchiectasis, pleurisy, and many other affections is compensated by the distention of previously healthy portions of the organ which occupy the vacuum produced by the contraction of the diseased portions and vicariously fulfil their functions. We shall return to this point later on.

Primary and compensatory emphysema together form a subdivision known as **vesicular emphysema.** It is distinct from interstitial emphysema, which has an entirely different pathogenesis, for the air escapes from the air-passages through injuries in the walls and forces its way into the lymph channels and clefts in the connective tissue of the lung, and sometimes even further into the mediastinum, and into the tissues of the body. A short section will be devoted to the discussion of this form.

We owe our knowledge of emphysema to Laennec, who described the disease and pointed out its characteristic symptoms, and who also advanced a theory in regard to its pathogenesis. Laennec was followed by Andral, Bouillaud, Louis, Lombard, and Woillez, who completed the symptomatology and morbid anatomy as far as clinical methods current in their time made it possible. Gavarret, Donders, Jenner [Gairdner.—Ed.], Skoda, and Hutchinson added their quota to the common stock of knowledge, which has been still more extended by the numerous modern authors whose names will be quoted in the following pages.

PRIMARY EMPHYSEMA.

MORBID ANATOMY.

When the thorax, which in pronounced cases of emphysema presents a characteristic form (see Symptomatology), is opened, the lung, instead of being collapsed, is found protruded; it appears distended with air; the edges, which normally are sharply defined at the mediastinum and diaphragm, are rounded and in contact in front from side to side, while the pericardial sac is almost or completely covered. The diaphragm is lower than in a normal subject.

After removal the lung appears enlarged, has a peculiar cushiony feel ("feather-pillow"), the surface is pale and often marked with peculiar stripes of pigment. On closer examination the alveoli are recognized as minute vesicles distinctly visible with the naked eye. In many cases the size of the alveoli is considerable, and emphysematous bullæ the size of a pigeon's egg have been observed. Bullæ of this magnitude are, as a rule, isolated, or two or three of them together form a conspicuous group. Their commonest seat is on the anterior margin of the lung, and they there appear even pedunculated or constricted. They do not always collapse when cut with a knife, because the walls are thickened and rigid. When a lung possesses numerous bullæ of this kind, as happens in exceptional cases, the term "batrachian lung" is sometimes used.

Vesicles the size of the head of a pin and over are always formed by the coalescence of a number of distended alveoli and passages leading from infundibula. The areas where the vesicles are most perfectly developed are usually conspicuous by the absence of pigment.

Diffuse emphysema never involves all portions of the lung to the same degree. Although in pronounced cases every portion of the pulmonary area is involved, the distention of the alveoli is always greatest at the margins and at the apex. Next in order of involvement is the surface, while the interior portions resist longest and show the least amount of pathologic change. It is also stated that emphysema is usually more extensive on the left, than on the right side; and that the base, especially of the right side, is more extensively involved, perhaps on account of the absence of the counterpressure of the liver on that side.

At all events, the apex and the anterior border of both lungs are to be regarded as the seats of predilection. There are, however, many exceptions. Emphysema may be confined to one lung; in such a case the diseased lung only is enlarged, and causes displacement of the heart and mediastinum toward the other side; in other cases the emphysema is confined to a single lobe. Again, the picture may be modified by the resistance of neighboring structures. A good many interesting things still remain to be discovered by some careful observer, but up to the present time little attention has been devoted to such refinements; indeed, the study of the whole subject of emphysema has been much neglected. Primary or diffuse emphysema merges into compensatory or partial emphysema by numerous gradations—there is no sharp dividing-line between the two forms. This is necessarily the case in practice. When the independent, characteristic features of emphysema begin to fade, and other symptoms assume greater prominence, the condition ceases to be described as emphysema, or emphysema is recognized merely as a concomitant phenomenon.

A senile emphysematous lung is distinguished by the fact that it appears contracted instead of enlarged, because it is frequently due chiefly to atrophic processes. Accordingly **senile emphysema** has been described as a separate disease, distinct from ordinary primary emphysema.

The emphysematous lung has been made the subject of numerous microscopic examinations. The most conspicuous feature is the enlargement of the alveoli and the atrophy of the septa which separate them. The latter may disappear altogether, so that several alveoli coalesce to form a single cavity; the boundary-line between alveoli and infundibuli becomes quite indistinct and it is impossible to tell where the former begin and the latter end. While one observer describes a structure as an alveolar space, another one will perhaps call it an infundibulum. Perforations appear in the walls that separate the infundibuli from the alveoli, and, indeed, the walls may disappear altogether in severe cases.

The discovery that a healthy lung possesses in the walls of its alveoli stomata which establish a communication between them is of some importance in connection with the pathogenesis of these perforations. These pore-canals, which were first observed by Kohn and later by Hauser,* who, again, showed them to be normal structures, and finally demonstrated by Hansemann† by means of injections, were recognized by numerous later authorities, so that their existence is now no longer a subject of controversy. The openings in the septa that have long been known to exist in emphysematous lungs‡ are now regarded by the authors as enlargements of already existing pore-canals, although it is possible that some of them may have been produced directly by some disease of the tissues.

The blood-vessels have been studied in a variety of ways. Isaaksohn§ appears to be the first who worked with the injection method. In the normal state the wall of the alveoli is covered by a dense, tortuous capillary network which extends into the lumen of the alveolus. In emphysema the capillaries are confined to the surface of the alveolar walls; the tortuosity disappears; and in places the capillaries are not even permeable for the injection material, a granular mass, the remains of thrombus formations, occluding the lumen. The production of these thrombi is declared by Isaaksohn to be due to changes that begin in the endothelium of the capillaries. The characteristic boundary-lines of the endothelial cells no longer appear when the tissue is injected with a silver solution; in places the walls are rough and granular, and covered with white blood-cells which become adherent to them and form the starting-point for the occlusion that develops subsequently. All authors agree in describing a dilatation of the capillary loops, with elongation and attenuation of the capillaries, either because they regard such a change as the necessary result of the dilatation of the alveoli, or because they have directly observed it in the specimens. Among the latter are Eppinger, and Klaesi, who says: "The atrophy of the capillaries is secondary to degeneration of the epithelium. The capillaries lose their support and are more exposed to traction and other injuries consequent upon the increased pressure and distention of the alveoli, because they are held fast only by a very thin membrane. The traction and distention on the capillaries tend to reduce the lumen to a minimum. In the end the constricted lumen, which for some time has been impermeable to morphologic elements, becomes interrupted, the walls come in contact with one another, degenerate, and disappear." He evidently saw no thrombus such as Isaaksohn describes, and refuses to admit a capillary thrombosis. Eppinger speaks of distention and attenuation of the vessels, and states that they are already obliterated at the central point. He assumes that the vessel becomes torn and leaves a small stump on each side of the point of division. The laceration of the vessels, he says, is sometimes, although rarely, accompanied by the production of small hemorrhages, because by the time ulceration takes place the vessels have already become obliterated to a great extent. He failed to see thrombi and does not believe that they are produced, on the ground that the distention of the vessels takes place very gradually.

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* *Ziegler's Beiträge*, vol. xv, "Ueber die Entstehung des fibrinösen Exsudates."

† *Berliner klin. Wochenschrift*, 1899.

‡ Ruysch, "Minima quaedam foraminula per interiorem faciem hiantia."

§ *Virchow's Archiv*, vol. LIII.

In view of the intrinsic importance of the behavior of the pulmonary capillaries, a reproduction of the excellent preparations by Isaaksohn in "Virchow's Archiv" are here given (Fig. 11). One of the pictures represents an injected specimen of the capillaries of a normal alveolus; the other shows the extreme degree of atrophy observed in the vessels as the result of emphysema. Since the vesicular network of the pulmonary capillaries evidently becomes constricted, and the meshes are widened out so that less blood passes than previously, the number of red blood-corpuscles that take up oxygen is more and more restricted and the breathing is necessarily impaired. The questions naturally suggest themselves: Is there disturbance in the minor circulation? Is there no obstacle to the action of the right heart? These questions have often been answered in the affirmative, and it has been thought that a congestion takes place which explains the hypertrophy of the right ventricle. There is no question that nature even in this condition finds a way to bring about compensation. If the blood is unable to pass in sufficient quantity through the alveolar capillaries, it forces its way through accessory channels, such as exist to a limited extent even under normal conditions, and under the influence of the sudden necessity become enlarged and assume a greater functional importance. Rindfleisch mentions such accessory channels. No exact investigations on the subject are, however, extant. But in any case it offends my notions of biology to assume that an important vascular region should simply become diminished in size. The circulation unquestionably undergoes profound changes; new communications are opened up; but can this be regarded as an abnormal process, sufficient to account for the constant development of hypertrophy of the right ventricle? I must confess that I am loath to accept such a theory. Compensatory changes of this kind, requiring a long period of time for their completion, do not imply any great expenditure of effort at any given moment. It has to be borne in mind that the capacity of the right ventricle is adjusted to a flexible amount of work, and that even under normal conditions the variations are considerable. But in time of disease, when man is warned and even compelled by nature to economize his strength, is it to be thought that the work of the right ventricle is increased to such an extent as to produce a permanent hypertrophy? I do not believe that the matter is as simple as all this. I am more inclined to ascribe the hypertrophy of the right ventricle, in large part at least, to the diminished aspirating power (negative pressure) of the lung. This aspirating power diminishes with the elasticity, and materially assists the onward movement of the blood as it becomes more and more feeble. A permanent and material increase of work is thus thrown upon the heart, and I incline to lay much greater weight on this event than on the disappearance of the capillaries.

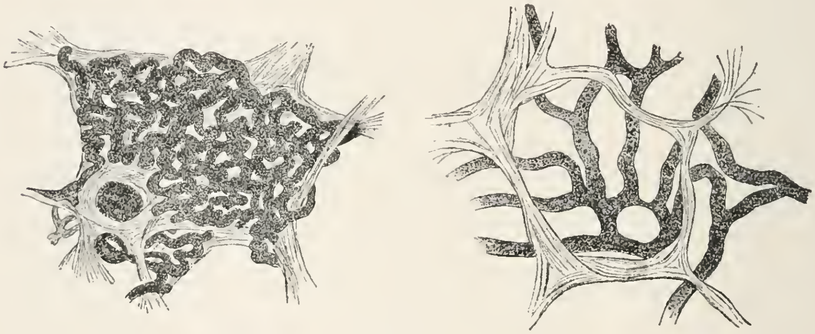
FIG. 11.

The distention of the alveoli is, of course, accompanied by changes in the endothelium, which undergoes fatty degeneration and pigmentary infiltration. In this connection the question of the normal condition of the alveolar epithelium has become the subject of a heated dispute among the authorities. It is now admitted that under normal conditions the alveoli possess a continuous epithelium consisting in part of large nuclear cells without protoplasm, and in part of small protoplasmic cells—so-called fission cells (*Spaltzellen*). In emphysema, however, the epithelium in the intervals between the capillary loops is interrupted, and may even be totally destroyed.

Biermer asserts that the epithelial cells undergo fatty degeneration. Klaesi* found a multiplication of the cells and saw signs of segmentation. He concludes from numerous preparations that the epithelial cells suffer a loss of their cement substance, become separated from one another, detach themselves from the capillary wall, and finally degenerate into granular masses. He insists that the capillaries at first remain intact, and that the disappearance of the capillaries is completed only after the degeneration of the epithelium has taken place. Eppinger, while unable to demonstrate that the epithelial cells became detached from the capillary walls, speaks of displacement or overlapping and fatty degeneration.

According to Rindfleisch, the smooth muscle-fibers found in considerable numbers in the walls of the infundibula undergo hypertrophy and form bundles of considerable size. The alveoli do not contain smooth muscle-fibers. If such have been described, it is probably because connective-tissue nuclei in the stroma were mistaken for them. Klaesi failed to find them in the walls and in the trabeculæ of emphysematous bullæ. The connective tissue of the alveoli is universally described as atrophic; it evidently shares in the pathologic process to the same extent as the rest of the tissue. Klaesi found specimens in which the connective-tissue was granular and pierced by clefts; Rainey distinctly speaks of fatty degeneration. The proliferation of connective tissue about the blood-vessels, which was assumed by Klob to take place, was not observed by any other investigator. Eppinger says it is always atrophic and in part undergoes fatty degeneration, as may be inferred from the granular nature of the ground-substance surrounding the gaps in the tissue. The same author, however, describes hypertrophy in the interstitial connective tissue.

Considerable interest has been aroused by the nuclei of the connective-tissue cells (the so-called stroma nuclei). In Germany Wagner† and his disciple O. Bayer‡ are at present engaged on the investigation of this question. The latter was able to convince himself that the number of these nuclei is not, as a rule, increased. In France no less an authority than Villemin§ has devoted his time to this study.

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* *Virchow's Archiv*, vol. CIV.

† Uhle and Wagner, "Handbuch der allgemeinen Pathologie."

‡ *Archiv der Heilkunde*, XI, p. 360.

§ *Archives générales*, 1866, II, p. 566.

The nuclei, according to his observations, hypertrophy to such a degree as to compress the capillaries. The latter then undergo fatty degeneration, break down, and are finally converted into sputum. It is to the loss of the capillaries that he attributes the openings that we now believe to be due to the pore-canals.

Klaesi regards these connective-tissue nuclei as lymph-corpuscles, and states that they sometimes congregate to form large masses; in addition to these lymph-cells he also saw a few smaller, almost homogeneous, structures. He regards these as the nuclei of the ground-substance. Biermer suggests that Villemin was deceived by epithelial nuclei.

A similar diversity of opinion exists in regard to the elastic fibers of the emphysematous lung. Since the elasticity of the lung is evidently diminished, it seems only natural to expect to find marked changes in the elastic fibers. Eppinger devoted considerable time to this subject, and found that the coarser fibers of the alveoli are forced apart and greatly reduced in size, while the finer ones disappear in many places. In advanced stages he discovered lacerated and split elastic fibers in the alveoli, a finding that has been confirmed, especially by French authors. Marfan, in his "Traité de médecine," volume IV, page 448, has an illustration of a bundle of lacerated elastic fibers coiled up in the shape of a corkscrew.

The laceration of delicate elastic fibers is regarded by Eppinger as the cause of the formation of minute clefts in the midst of the inter-capillary spaces, which are bounded by a small amount of ground-substance or directly by a mesh of the capillary network. The larger, secondary clefts are bounded by numerous capillaries giving off small branch-like processes which project into the cleft and end blindly. They are probably torn capillary vessels. Eppinger was not acquainted with the normal pores that exist between the alveoli, and it is probable that some of these at least, in a dilated condition, correspond to his clefts. It is not impossible, however, for clefts to be produced in other places, independently of the pores.

German authorities that followed Eppinger failed to confirm the presence of split elastic fibers; Klaesi sought for them in vain. The latter is not inclined to admit a true atrophy of this extremely resistant tissue, and believes, rather, that there is an overlapping and displacement of the fibers which might simulate atrophy. On the other hand, a number of authors speak of the disappearance of the elastic tissue, as, for example, Birch-Hirschfeld,* while Ribbert † does not share this view, believing that elastic fibers can readily be demonstrated in every portion of the emphysematous tissue. But this guarded statement tells us no more than that he does not consider the question settled. The latter opinion is evidently shared by Hansemann, who recently inspired his disciple Sudsuki ‡ to prepare another arbeit on the elastic fibers in emphysema. As a result of his investigations Sudsuki finds that there is neither a qualitative nor a quantitative diminution in the development of the elastic fibers, and that they *do not disappear any earlier than the other tissue elements in this disease.* They therefore simply suffer along with the rest of the tissue. He disagrees with Eppinger emphatically, and believes that the split elastic fibers described by the latter depend on an error in observation. In compliance with my request, Professor Spalteholz, who formerly devoted a good deal of time to the various methods of staining elastic fibers, has had a number of specimens from a case of severe emphysema prepared according to the newest staining methods (Weigert's and Spalteholz's own methods). In these preparations he was unable to demonstrate the presence of lacerated or split elastic fibers. The differences noted on comparing the specimens with a normal lung were not conspicuous, and can be perfectly well explained by a different degree of distention of the pulmonary tissue at the time of fixation.

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* "Lehrbuch der pathologischen Anatomie," vol. II.

† "Lehrbuch der pathologischen Histologie."

‡ *Virchow's Archiv*, vol. CLVII, p. 438.

The absence of pigment from the emphysematous portions of the lungs is admitted by all the authorities; no exact investigations of this point have, however, been made. Grawitz relates that in his case of hydropic emphysema the pigment was washed out of the greatly dilated lymph channels of the connective tissue; and that in inflammatory and atrophic emphysema the pigment, after the connective tissue has been converted into cells, passes along with the latter into the lymph channels and is thus carried off.

The differences in the morbid anatomy of senile or atrophic emphysema and ordinary emphysema depend on the uniform contraction of the lung (small-lunged emphysema of Jenner). It is also a diffuse emphysema involving both lungs. Absence of pigment is the rule, but cases also occur that are characterized by enormous deposits of pigment. The smaller bronchi are regularly dilated.

ETIOLOGY.

The first thing that strikes the observer when he examines an emphysematous lung is the failure of the organ to contract as in health. When the thorax is opened, the lung retains its distended condition, while a healthy lung collapses. It is evident that the elastic tissue has lost the requisite strength to regain the normal volume. This absence of elasticity, which forces itself on the attention at the autopsy table, has been regarded as the basal cause of the disease. The first question that is asked in the inquiry after the cause of emphysema is, Why do the lungs lose their normal elasticity? If a rubber band is stretched too long, and beyond a certain degree, it gradually loses the power to regain its original volume; and, applying this experiment to the lungs, it is concluded that frequent and excessive distention is the cause of emphysema. This argument, while simple enough, is anything but convincing. It is quite evident that emphysema might be produced in this way, but there is nothing to prove that it must be so produced. If the elastic fibers are injured by any cause, they lose their normal degree of elasticity. It is not at all necessary that the injury should be produced by overdistention; furthermore, it is an arbitrary assumption to say that the injury of the elastic fibers is primary. Although it cannot be denied that it is a necessary factor in the development of emphysema, that does not prove that it is the primary factor. In attempting to define the causes of emphysema it is therefore incorrect to investigate exclusively the cause of the loss of elasticity. I prefer for the present to view the matter from a clinical standpoint, and therefore inquire under what conditions emphysema, the *disease* with which we are now dealing, develops.

Emphysema has been designated a hereditary disease, and writers have indulged in a great variety of speculations on this point. The family histories have been ransacked, and it has been found that in the case of more than 50% of emphysematous subjects emphysema could also be found in the parents (I purposely avoid the word demonstrated). Jackson* instituted a comparison between 28 emphysematous subjects and 50 non-emphysematous subjects; in the case of the former he found 20, in the case of the latter 3 emphysematous relatives in the preceding generation. These statistics do not, of course, bear criticism, but there is no harm in quoting them.

It has also been pointed out that emphysema is not the only condition to be looked for in the preceding generation, and that chronic rheumatism, gout, obesity, hemorrhoids, gravel disease, nervousness, are to be considered as hereditary factors capable of determining a more or less pronounced predisposition to emphysema. But no causal relationship between the diseases enumerated and emphysema has ever been demonstrated. It is evident that speculations of this kind only tend to confuse by seducing the speculator into the pleasant realm of high-sounding phrases, which courtesy demands shall be accorded the euphemistic name of scientific hypotheses. Such speculations merely show that children of healthy parents have better prospects of remaining healthy than the children of unhealthy parents, a result that can be arrived at without any very profound investigations.

In examining the literature, occasional case-histories are met in which the physician noted an astonishing number of cases of emphysema in the same family, and the assumption of hereditary predisposition is largely based on observations of this kind; but as a matter of fact they are relatively infrequent. The hereditary factor cannot be regarded as playing an important rôle.†

It may be assumed that the elastic tissue possesses greater firmness in some lungs than in others, and the latter may be considered to possess a predisposition to emphysema. For example, we may indulge in the general statement that an infantile lung, owing to its more delicate structure, is more likely to become emphysematous under the influence of appropriate injuries than are the lungs of a more vigorous period of life. But, on the other hand, it possesses the advantage of all infantile organs in the greater tendency to recovery from disease; and, as a matter of fact, acute emphysema following whooping-cough or diphtheria often ends in complete recovery. In later periods of life—beyond the thirty-fifth year—the constantly increasing debility and diminished tendency to recovery characteristic of old age make themselves felt more and more. These things are all perfectly simple, and necessarily follow from the nature of things, and have not as yet been shown by scientific investigation to have any special bearing on emphysema. Nevertheless they are, according to the present state of knowledge, sufficiently well established *à priori* to be worthy of acceptance as long as nothing is brought forward to overthrow them. So far as is now known, there is no disease that needs to be especially looked for in the parents except emphysema itself. An exception might be made in favor of gout* (see under Complications). Cases of direct heredity might be cited; but we must repeat that there is a dearth of well-authenticated observations bearing on the question.

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* Waters, *Archives générales*, 1864, vol. II, 594.

† Hertz in Ziemssen's "Handbuch"; Schnitzler, *ibidem.* Schmidtlein, "Deutsche Klinik," 1864, p. 214, and other authors.

The next etiologic factor after heredity is the **age**. During the period of development the subject is more susceptible than during middle age, because the tissue elements are less robust and possess a lower power of resistance. Accordingly a rapid or even sudden development of pulmonary distention is not infrequently observed in childhood under the influence of some acute injury; but true chronic emphysema, in the narrow sense of the word, is extremely rare. The great elasticity of the parts and their power of metamorphosis make recovery possible where it would be out of the question in adults. Genuine chronic emphysema usually does not occur before the third decade, and after that becomes more and more frequent as age advances. Extreme old age is especially predisposed to emphysema. because the cartilages often become rigid. I say often advisedly, for it is not uncommon to observe perfectly normal expansion of the thorax in men between sixty-five and seventy-five years of age, and at the autopsy the cartilages may be found entirely free from calcification. To regard age in itself as the cause of calcification I believe to be erroneous; but the injuries that lead to calcification in many individuals tend to accumulate in the course of years, so that calcification becomes so extreme as to constitute a distinct obstacle to the movements of the thorax. When this condition has developed, the thorax is unable to expand properly during inspiration or to collapse during expiration, and an extra amount of work is thrown on the diaphragm. This the latter is undoubtedly able to perform, providing the individual does not attempt to exert himself as much as a younger man; and plenty of old people with rigid cartilages are quite free from emphysema. Freund's theory of a primary disturbance of nutrition in the ribs as a cause of emphysema may be explained in this way; and there is no need to set up an idiopathic disease of the costal cartilage as a cause of emphysema. Cantani, in assuming a specific catarrh as the cause of emphysema, and Freund, in erecting his specific ossification of the ribs, are equally extravagant; but the latter's explanation of the condition ought not, on the other hand, to be condemned altogether. The importance of the elasticity of the thorax in the act of respiration has been too much neglected in favor of the more apparent and more easily demonstrable influence of the diaphragm; but of that later. In this connection a paper by Huttkrantz* strikes me as very interesting. He made a comparison between the diaphragm and the thorax in regard to their respective shares in respiration by inclosing the lower part of the body and the trunk, with the exception of the head, separately in an air-tight metal vessel provided with a water manometer. He concludes from his experiments that of the 490 cubic centimeters of inspired air, 320 are accounted for by the expansion of the thorax, and 170 by the depression of the diaphragm. The influence of the depression of the diaphragm, as determined by this method, may, it is true, be somewhat less than it is in reality, because the air in the abdomen suffers compression at each inspiration, but in spite of this source of error the experiments clearly demonstrate the great importance of the thoracic movements.

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* Greenhow, *Lancet*, 1867.

When the thorax has lost its elasticity, the corresponding amount of space is lost; for the elevation which the inspiratory muscles are capable of bringing about can only be utilized by the movement of the diaphragm. It follows, therefore, that any disease that leads to rigidity of the thorax must bring about an injury of this kind. Such a disease materially interferes with the expansion of the upper portion of the lung. In persons with a tendency to arteriosclerosis the conditions for calcification of the costal cartilages are especially favorable, and this is probably the reason why arteriosclerotic subjects are so commonly found to be emphysematous. The arteriosclerosis unquestionably also affects the circulation, but it would be wrong to suppose that it exerts its influence only through that channel. Circulatory disturbances manifestly bring about unfavorable conditions for the expansion of the lungs. The suction-power of the heart is lowered, the movement of blood in the capillaries is retarded, a tendency to bronchial catarrh is thus encouraged, and injury is added to injury, with the result of senile emphysema. The latter should therefore never be regarded as a simple senile phenomenon of involution.

A sharp line of distinction is often drawn between senile emphysema and ordinary chronic emphysema, and the former is frequently treated as a distinct disease. As a matter of fact, however, ordinary emphysema very often gradually merges into senile emphysema as the individual grows older. The two forms cannot be strictly differentiated.

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* *Med. Centralblatt*, 1890, p. 226.

The next etiologic factor is the **sex**. A general impression prevails that emphysema is more frequent in men than in women.* The difference is readily understood, as men are much more engaged in occupations that predispose to catarrh, and the great majority of them are addicted to the abuse of alcohol, which, in turn, by weakening the heart, leads to disturbances in the lungs and brings about chronic hyperemia of the bronchi.

Occupation.—The occupations most to be dreaded are those that favor the development of chronic bronchitis through the inhalation of dust, or even produce severe inflammatory disturbances. (See Pneumonokoniosis.) In the second place, occupations necessitating repeated and excessive exertion of the lungs with an increase of air-pressure. Among the latter the most important is the playing of wind-instruments, because the free circulation of the air is interfered with by the obstacle applied to the mouth. It is to be noted, however, that it is possible with care to play a wind-instrument in such a way as not to injure the lungs, and our orchestras contain plenty of perfectly sound cornet players. Of course, these men must be careful not to overtax their lungs and not to begin playing with their accustomed vigor too soon after a debilitating disease. It is not difficult to understand the case of the trumpeter who developed emphysema of an entire lung which had previously been infiltrated with pneumonia. The same conditions are found among glass-blowers and plumbers who use the blow-pipe. An expert blower who is otherwise healthy can follow his trade without injury to his health.

As a curiosity it may be mentioned that the raising of heavy weights, the effort incident to labor and to defecation, violent exercises with the arms, or even a sedentary mode of life, and, finally, singing (!) have been regarded as predisposing factors. There is no doubt that under especially unfavorable conditions they might all exert an injurious influence, but to vindicate such a statement the onset and the duration of the injury must be accurately determined and investigated. In the cases in which I have found causes of this kind cited, it appeared to have been done either for the sake of saying something, or it was as a mere subterfuge on the part of untrustworthy individuals who wished to hide the true cause—namely, their fondness for drink. Nevertheless they are religiously noted down in the text-books.

Among actual diseases, bronchitis and asthma occupy the first place; and all diseases of the air-passages that lead to bronchitis and asthma are also mentioned as predisposing factors. Bronchial catarrh with watery, mucopurulent sputum that is easily coughed up, is regarded by Traube as a distinct exception; such a condition is possible, but its occurrence is not proved by reliable case-histories.

As a rule, the consistency of the secretion in chronic catarrh is very variable; sometimes it is watery and easily expectorated, at other times it is tough and requires more and more violent cough for its removal. Cases of acute bronchorrhea occur only in bronchiectasis and putrid bronchitis. If there is bronchitis, other pathologic changes are present, which, again, may predispose to emphysema. The conditions then become extremely complicated.

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* Hertz gives the proportion of incidence as 147 in men to 42 in women.

Traube* insists that two forms should be distinguished: "A diffuse form of catarrh with homogeneous mucopurulent, nummular, floating expectoration, characterized by the auscultatory phenomena of coarse, bubbling râles in the postero-inferior portions of the thorax, does not predispose to emphysema no matter how great the dyspnea associated with it.

"On the other hand, it cannot be denied that a predisposition to emphysema resides in the form described by Laennec as dry catarrh. In this affection. . . . (here follows a description of asthmatic sputum †). The auscultatory sign in dry catarrh is a sibilant râle, which therefore also indicates a contraction of the smallest bronchi. During the act of coughing a condensation of air takes place in the entire respiratory tract, and wherever the smallest bronchi are temporarily occluded by the 'cylindric corpuscles' so that no air can enter, extreme distention must take place, and thus lead to dilatation of the portion of the lung supplied by these bronchi.

"In many cases of emphysema small atelectatic areas are found, which are not, as Rokitansky assumes, caused by compression of the pulmonary tissue, but by a permanent hermetic closure of the smallest bronchi. In cases of chronic pulmonary tuberculosis the sputum often consists of villous masses containing a tangle of delicate yellowish-white threads. In these cases also the above-described mechanism, resulting in the production of emphysema and atelectasis, is manifested."

The distinguished clinician, in harmony with the opinions prevalent in his time, made many assertions that we have ceased to admit at the present day. The unfavorable effect of cough in constriction and occlusion of the bronchi is, however, as true to-day as it was then; it is also true that certain forms of bronchial catarrh do not predispose to emphysema at all, while others, especially the asthmatic forms, do predispose to emphysema in a very high degree. But the forms which are included in the first group are not sufficiently characterized by describing them in this way. It is impossible to set up two categories, except for purposes of didactic teaching. As a matter of fact, there are a thousand gradations and innumerable conditions, transitional forms, between those catarrhs that lead to emphysema and those that do not. At one end of the series may be placed asthma; at the other, bronchiectasis.

Next to catarrh are all those diseases that tend in any way to narrow the lumen of the air-passages. Stenosis in this connection must be clearly distinguished from *occlusion*. There is a degree of stenosis that practically amounts to occlusion. If this stenosis is in the large air-passages, a general emphysema may result. This occurs in goiter; in the presence of neoplasms; ulcers; compression; cicatrization; disease of the glands in the trachea and main bronchi; laryngeal diseases attended with stenosis and alterations in the pharynx, such as enlarged tonsils and the presence of pharyngeal tonsils. If the stenosis is situated in the smaller branches, emphysema will develop only in those portions of the lungs which they supply. It would be a mistake, however, to assert that stenosis in itself is necessarily followed by emphysema. In addition to stenosis, there must be some condition by which the circulation of air in the bronchial tree suffers some special interference. This may often be the case, but is not necessarily always the case. The question will be discussed later.

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* "Ges. Abhandlung," vol. II, p. 980.

† One of the earliest references to the spirals is here found; Traube calls them "cylindric corpuscles."

Since it must be accepted as a well-established fact that certain nasal diseases, such as exostosis of the septum, hypertrophy of the turbinates, polypi, and hypertrophy of the pharyngeal tonsil, predispose to asthma, it is equally certain that the same conditions may lead to distention of the lungs, and ultimately to emphysema. Sandmann* has described a number of cases in which dilatation of the lungs was improved or even disappeared (?) after the nasal obstruction had been removed.

Cervello† experimented on dogs by sealing up the nasal orifices, and observed distention of the lungs after a few days.

Sticker's‡ theory of a causal connection between atrophy, or dry catarrh of the nose or pharynx, and emphysema deserves to be mentioned. This form of catarrh may extend to the trachea and even further down into the smaller bronchi. Is this merely another case of emphysema being caused by catarrh, with the simple difference that the special form of catarrh begins in the nose? Sticker goes still further, and erects under the name of xerosis of the mucous membranes a disease which, he says, leads to wide-spread but moderate atrophy of all the mucous membranes of the body and, as age advances, to an increasing enlargement of the nasal chambers, the pharyngeal cavity, the larynx, the trachea, and finally the lungs. Friedrich also is inclined to adopt this view. He writes as follows §:

"In cases of marked atrophy with pronounced ozena of the nose and pharynx, experience teaches us to expect not only chronic bronchitis, but also pulmonary emphysema and asthma-like attacks. If such a condition is met in elderly persons who have all their lives suffered from chronic bronchitis due to ozena, it is readily explained as senile emphysema or as a secondary emphysema, such as may develop gradually in bronchitis. But how are we to explain such cases of pulmonary emphysema in young persons barely twenty years old with all the symptoms—especially dyspnea and cyanosis—which are found only in the severest grades of emphysema? I remember particularly a healthy young farmer, twenty-one years old, who suffered from a severe emphysema, and the only explanation that could be found was a marked ozena which the patient said he had had for a long time. Such cases speak for the existence of a xerosis, after Sticker." If xerosis leads to emphysema, it can only do so in the same way as ordinary catarrh, for it does not produce any actual disturbances of the air movement. It is probably for this reason that Sticker states that his cases were so-called senile atrophy of the lungs described by Rokitansky and not true pulmonary emphysema. The possibility of primary senility, especially on a luetic foundation, is not to be denied; but in any case the literature collected by Sticker in regard to the causal connection between xerosis and emphysema is insufficient in quantity and untrustworthy in quality. Before the existence of a xerosis associated with emphysema or pulmonary atrophy can be admitted and properly described, more complete case-histories by trustworthy and critical observers are required. Such cases are not likely to be common. A small number of cases of ozena and atrophic rhinitis persisting for years without the development of emphysema occur in the practice of every busy physician.

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* *Berl. klin. Woch.*, 1888, No. 2.

† Abstract in *Klin. Centralblatt*, 1890, p. 885.

‡ *Deutsches Archiv für klin. Medicin*, vol. LVII.

§ "Rhinologie, Laryngologie und Otologie in ihrer Bedeutung für allgemeine Medicin," 14. ("Rhinology, Laryngology and Otology in General Medicine," Friedrich, 29–30. Saunders & Co., Philadelphia, 1900.)

A question that occupied the author's attention in connection with certain cases of eosinophile catarrh is whether **syphilis** plays any considerable part in the etiology of emphysema. The author has several times had syphilitic individuals present themselves at the public clinic for very obstinate eosinophile catarrh, which was promptly and permanently cured by potassium iodid. He therefore feels quite certain that there are eosinophile catarrhs that have nothing whatever to do with asthma, and regards it as probable that some of these cases rest on a syphilitic foundation. Inasmuch, then, as syphilis leads to eosinophile catarrh, it may also be said to predispose to emphysema.

The diseases that lead to violent dyspnea without directly affecting the air-passages may now be considered. Again, the dyspnea must be such as to interfere with the circulation of air, and the injury must be such that air can enter more freely than it can escape. This condition is fulfilled in many dyspneic attacks. A brief attack, as a rule, leads to acute emphysema; but whether it shall be acute or chronic depends not only on the duration of the injury, but also on the resisting power of the lungs. In old persons a comparatively short attack may leave a permanent emphysema. Few trustworthy observations bearing on this point could be found. Koranyi tells in his article of a woman who had an embolism in the main branch of the pulmonary artery, and four years later died with symptoms of intense dyspnea; emphysema was found at the borders and in the lower lobes of both lungs. The case is not conclusive, as the previous condition of the lungs is not known. Embolism of the pulmonary artery has long been known as a cause of acute emphysema. Among other causes that act in a similar way are the entrance of air into the veins; the inhalation of asphyxiating gas, such as carbon monoxid, hydrogen sulphid, and sewer-gas; the inhalation of irritating gases, such as chlorin, ammonia, and hot vapors; the entrance of foreign bodies into the air-passages; respiratory disturbances in diphtheria and whooping-cough. But the acute distention of the lungs produced by these causes very exceptionally leads to chronic emphysema.

The same is true of attacks of stenocardia and cardiac asthma. The latter will have to be regarded as a symptom of emphysema, but cannot be conceded to play any considerable part in the etiology.

Among the diseases outside of the lungs that are regarded as predisposing to emphysema, pleurisy with adhesions of the two layers of the pleura may be mentioned. The normal lung makes a considerable excursion downward and forward at each inspiration. The anterior and inferior portions of the lung are displaced, and the space is occupied by parts coming from behind and above. In the presence of adhesions the amplitude of the movements of the lung depend altogether on the comparative elasticity or rigidity of the adhesions. The diaphragm unquestionably moves downward; if the lung is unable to follow in the normal way, the bronchi unquestionably become distorted and compressed in places, and certain portions of the lung, in their effort to fill the vacuum, become greatly distended. The adhesions, however, rarely become so extensive as to produce a general emphysema. Respiration is constantly carried on and offers a strenuous resistance to the formation of excessive and injurious adhesions. It is in this way that partial or compensatory emphysema is no doubt to be in great part explained. When the adhesions are robust and very extensive, the heart also becomes involved and the circulatory disturbances are more conspicuous in the clinical picture than the emphysema.

Diseases of the heart itself, such as myocarditis, valvular lesions, and synechiæ, are frequent causes of emphysema. If the circulation in the bronchial tree or in the alveoli is impaired, it is natural to hold a congestive catarrh responsible for the condition. Next there are the changes in the blood-vessels, especially arteriosclerosis of the coronary vessels, the arteriosclerotic changes so frequently found at the points where the bronchial arteries are given off, and, finally, similar alterations of the pulmonary artery and its branches. These are the cases that Grawitz has described under the name of edematous forms of emphysema, and in which the circulatory disturbances must be recognized as the basal lesion. This author is nevertheless of the opinion that even in these cases the development of emphysema cannot be explained without the intermediate link of a disturbance to the ventilation.

A number of general disorders characterized by a special tendency to involvement of the air-passages must be considered in the etiology of emphysema. **Rachitis** almost regularly leads to violent and persistent bronchial catarrh. If the disease seldom appears in the list of etiologic factors, it is no doubt owing to the fact that pulmonary alterations usually undergo favorable involution after the rachitic process has been arrested.

Alcoholism is a much more important etiologic factor. It is absolutely certain that the great majority of emphysematous subjects among the lower classes are recruited from the ranks of alcoholics. Cold is generally alleged to be the cause, but the true offender is alcohol. It keeps up a chronic catarrh not only because it is largely excreted through the mucous membranes, but also by impairing the action of the heart until it is inadequate to maintain a proper circulation in the lungs. Ultimately alcohol undermines the general nutrition and power of resistance of the body: thus one injury is added to another, with the final result that emphysema becomes one of the commonest diseases treated in public clinics.

The abuse of tobacco has also been accused of producing emphysema, and no doubt with some reason, especially as it is so frequently combined with alcoholism.

Can emphysema be due to purely **nervous causes?** An acute distention of the lungs occurs in conditions of terror in the insane * and subsides along with the other symptoms. Ziertmann explains the occurrence as the effect of central irritation of the vagus in the medulla oblongata; it is not known whether a chronic emphysema develops on the same basis.

A number of other diseases are mentioned in the literature, and no doubt they harbor certain general injurious factors, but do not directly affect the air-passages and particularly the lungs. Dyspepsia, dilatation of the stomach, and the like conditions may be mentioned for the sake of dismissing them; they cannot be regarded as constituting a cause or even a predisposing factor.

More recently **traumatism** has been accorded a certain rôle in the etiology. Any injury affecting the body may ultimately become a predisposing factor. It is customary to say that the normal resistance of the body is weakened, and, that being admitted, it is quite easy to derive any other injury therefrom. This principle is exploited to its full extent by those who take advantage of the law to obtain damages or an annuity for disability. If a person develops emphysema after an accident, the accident no doubt is a contributory factor in its production—no one can deny that. The injury need not necessarily have affected the chest; it may be any other portion of the body, even a psychic influence is held to suffice. It is to be regretted that this cheap method of reasoning is so popular among physicians. It is no more than we could expect from the layman who is looking out for an annuity, but the argument has no scientific basis; strictly speaking, it is a mere commonplace. If a traumatism is really the cause of emphysema, it ought to be possible to demonstrate its mode of action scientifically; if this is impossible, it is the physician's duty not to repeat mere phrases that have come into fashion, but to enter a vigorous protest against them. It is, I repeat, to be regretted that the mischief owes its origin to the physicians. The commonest causes of emphysema, *chronic catarrh* and *alcoholism,* are most prevalent among the working classes. Whenever these people are treated for an injury, they are by that very fact brought into surroundings which counteract the true causes of emphysema. This undeniable fact is too often overlooked. Besides, the assumption that the general powers of resistance are diminished after traumatism is in itself incorrect. Every form of irritation is followed in the organism by a distinct reaction, and as long as the reaction does not exceed the limits that correspond to the functional capacity of the irritated organ, the powers of resistance are not in any sense diminished; on the contrary, an increase up to a certain point may be expected. It is neither *à priori* certain nor even probable that a diminution in the powers of resistance takes place. A careful search should be made for objective signs that would clearly prove such a diminution, but no one ever thinks of doing so. Any cheap hypothesis passes current for a scientific fact, especially if it is dished up with plenty of foreign words and quotations. It is so much the fashion to approve of our (German) social code that the danger of indiscriminate charity is too apt to be overlooked. This is not the first instance of good-natured weakness masquerading in the guise of humanity and producing incalculable harm. The jurists have lightly appropriated the phrase "diminution of the powers of resistance from injury," and many of them, like the doctors, consider it rank inhumanity to refuse to admit this "scientific truth" and all its consequences for the benefit of the laboring man. Such a tendency is fraught with the greatest danger to the dignity of the law, for ordinary common sense can never be trodden in the dust, and reaction is sure to follow sooner or later. *Videant!* (Time will tell.)

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* Ziertmann, *Münchener med. Wochenschr.*, 1894, 38, 39.

What common quality is there in all the numerous diseases that are regarded as predisposing to emphysema, leaving out those of the latter group which may occasionally predispose to emphysema, but are of course excluded from any scientific analysis? Why do they lead to emphysema? One thing is to be borne constantly in mind: they do so more or less frequently, but not with absolute constancy. If I were asked to name a disease that leads to emphysema, I should select chronic bronchial catarrh and, possibly, asthma, with the proviso that the disease last long enough. In practice, it is found that even these diseases are not absolutely certain to be followed by emphysema; but such a result is so frequent that the relation of emphysema to bronchial catarrh may be said to be not that of a special affection, but rather that of a terminal stage. The same is not true of all other diseases; they may lead to emphysema, but, again, the lung may escape no matter how long the duration of the disease in question.

It all depends on the fulfilment of the condition which has already been touched upon, and to which a few more words may now be devoted.

It is evident that any disease of the respiratory passages, of the lungs, of the heart, and of the corresponding nervous domains, may produce a more or less severe grade of the injury referred to (interference with the movement of air). But emphysema does not necessarily result, unless the normal escape of the air is in some way interfered with. If the entrance of air alone is impeded, atelectasis and hypostasis are the natural results. Anything that disturbs the relation between the escape and the entrance of air in such a way as to make the former more difficult than the latter, is bound to produce emphysema. If the escape of air is obstructed in circumscribed portions of the lung, there results partial emphysema, which is under consideration here; if the escape of air is obstructed in larger portions, or in the entire lung, diffuse or primary emphysema is the result.

We are now ready to ask the question: What conditions present themselves in practice that produce a diffuse emphysema of this kind?

It is at once evident that diseases confined to circumscribed areas or foci cannot produce such a result. Diseases of this kind are tuberculosis, carcinoma, whether metastatic or primary, pulmonary abscess, and gangrene. From their very nature it follows that they can at most produce a partial emphysema; for diffuse emphysema to be produced the effect of the lesion must be more or less general.

The causes of emphysema that have a scientific value have in common the property of disturbing the free movement of air in the bronchi, in the infundibuli, and in the alveoli, and of causing a certain degree of dyspnea. Marfan says: "The principal symptom of emphysema is dyspnea," but I go even further than he, and regard the dyspnea itself as an essential factor for the production of emphysema. Not every form of dyspnea, however; let us be quite clear about this. Whenever the movement of air in the respiratory passages is disturbed, dyspnea necessarily results; for normal respiration is accurately adjusted to the needs of the body for oxygen. If more oxygen is required, the breathing becomes deeper and more rapid; if less oxygen is needed, on the other hand, the respirations become fewer in number and more shallow. Acceleration and deepening of the respiration are performed automatically, and the proper maintenance of this relation is the surest sign that breathing is normal. When a man breathes more rapidly after running, we speak of it, it is true, as dyspnea, but it is really a very bad expression. More correctly it should be called *eupnea*. The body is doing what physiology requires it to do in the best possible manner. There is nothing morbid or morbifying about it, as long as the limits are preserved within which the healthy organism can increase its activity without injury to itself. These limits can be narrowed by overanxiety and by taking too good care of one's self, and, on the other hand, can unquestionably be enlarged by exercise and hardening processes.

If the free circulation of the air in the air-passages meets with an obstruction, the normal relationship between inspiration and expiration and between the depth and frequency of the respiratory movements is altered, and then a true pathologic dyspnea exists.

The position of emphysema as an independent disease is greatly strengthened if the causal condition periodically subsides, or at least sinks into the background, so that its symptoms become quite subordinate to those of the emphysema. The affection is then a true primary or substantive emphysema.

Among the diseases that have a wide-spread effect on the bronchi and on the lungs, croupous pneumonia and inflammation of the bronchi are the most prominent.

Of the former it cannot be said that it impedes the escape more than the entrance of the air; in fact, the direct opposite is the case; it is, however, true of bronchitis. Many authors ever since the time of Laennec have recognized the fact that mucus in the bronchi obstructs the entrance of air much less than its escape.

Since Riegel demonstrated this positively with the aid of the graphic method which he elaborated, the question may be regarded as finally settled. I shall therefore quote this important passage *in extenso* ("Athembewegungen," p. 113): "Even though these catarrhs (that is, chronic catarrhs extending to the finer bronchi), if they attain a certain degree of intensity, offer a certain obstacle to the entrance of air, it is nevertheless the escape that is chiefly impeded. It is therefore to be expected *à priori* that severe catarrh of the finer bronchi should intensify the above-mentioned symptoms of emphysema, and this is fully confirmed by examination, which demonstrates that the subsidence and exacerbation of the catarrhal symptoms are accompanied by a corresponding increase and decrease of the expiratory obstructions." Inspiration is an active factor; negative pressure makes itself felt with great uniformity in all portions of the bronchial tree; if there is an obstacle in any portion of the bronchial system, it alters neither the direction nor the point of attack of the negative pressure until it has been overcome. The influence of the striated muscles in quiet breathing cannot as yet be regarded as being positively established. It used to be customary to ascribe very little importance to the voluntary muscles, the lion's share being accorded to the elasticity of the lungs and of the thorax. Now, however, that Henke asserts that the position of equilibrium of the thorax coincides with inspiration, and that the thorax therefore has a tendency to return spontaneously to a position of moderate inspiration, a much more important rôle must be assigned to the internal muscles than has been done in the past, provided, of course, that his view is correct. We shall return to this point later. It is at least certain, however, that the muscles do not share in the respiratory movements to the extent of exerting pressure on the lungs, but they probably do assist in maintaining the proper harmony between the movements of the thorax and those of the lungs. In the presence of any pathologic process, which is necessary for the production of an abnormal pressure, the contraction of the muscles must, however, act in a number of different ways, none of which can be regarded as favorable. On the contrary, the effect is quite indeterminable and is unquestionably often unfavorable. By exertion of the inspiratory muscles the entrance of air can be facilitated without harm and without difficulty; whereas by exertion of the expiratory muscles the escape of air may be expedited, but may also, under many conditions, be interfered with. Therein lies the danger of voluntary and forced expiration, as well as of cough.

All forms of bronchitis are therefore dangerous, but especially those associated with the production of tenacious mucus and active expiratory efforts, such as the rheumatic forms and the bronchitis accompanying whooping-cough, and these, accordingly, are the main causes of emphysema. Other conditions that interfere with inspiration to the same degree as with expiration are, obstructions in the large bronchi, in the larynx, in the nose, and in the nasopharyngeal spaces, such as tumors, cicatricial contractions, and inflammations. They are, of course, much less likely to produce emphysema. Under certain circumstances they may, it is true, act somewhat like valves and facilitate one phase of the respiration while they obstruct the other. If, therefore, they interfere chiefly with expiration, they may also lead to emphysema. As these conditions are apt to induce a secondary irritation of the bronchi, they are objectionable chiefly because they keep up a chronic bronchitis which, as has been explained, acts as a frequent predisposing cause. If they interfere more with inspiration than with expiration, the tendency to emphysema is not thereby removed, for the possibility of catarrh being superadded is not excluded, although, of course, the condition may subside without emphysema being established.

Other diseases act in a similar manner. Thus, extensive pleural adhesions are constantly mentioned, but in this condition the relations are subject to great variations. Some adhesions are associated with great disturbance of the respiration, while others are practically insignificant, the differences depending on the firmness of the adhesions, as well as on their seat and on the degree to which the heart is involved. Theoretically it is quite easy to say that the adherent portions of the lung find themselves impeded in their efforts at expansion, while others expand irregularly and thus lead to the production of compensatory emphysema. But this is an unwarrantably simple way of looking at the question; adhesions frequently exist without producing any emphysema.

I am inclined to lay more stress on diseases of the heart. The frequency of heart disease in emphysematous subjects at once becomes evident if the condition is inquired for. The only question is, Which of the two diseases is primary? Hitherto it was always said that the emphysema was the primary condition, but that is going too far. The frequency of cardiac affections in emphysema is illustrated in the table prepared by Chambert.* Among 258 cases of pulmonary emphysema he found:

The heart muscle alone diseased in	30	cases.
The muscle and the valve diseased in	58	"
The muscle diseased and the aorta atheromatous in	15	"
The valves alone diseased in	18	"
The aorta alone atheromatous in	19	"

It is evident that valvular lesions are not produced by emphysema; but how is it with disease of the muscle, synechiæ, and atheroma? For my part, I consider these conditions also primary. The emphysema which follows is often only partial, although every gradation is found up to the diffuse; and the impossibility of drawing a sharp distinction between the various forms in practice is very evident, particularly as emphysema secondarily leads to hypertrophy and dilatation of the right ventricle, and therefore also indirectly brings about disturbances in the nutrition of the heart muscle. It is anything but easy to determine in a given case whether one has to deal with a cardiac affection, with secondary emphysema, or with an emphysema with secondary cardiac affection. The older views, according to which emphysema was always regarded as the primary disease, were much too one-sided. At the present time there is a gravitation toward myocarditis, and it will no doubt result in going to an extreme in the other direction.

Among the forms of catarrh concerned in the etiology of emphysema I have included congestive catarrh. According to the investigations of Basch and his disciples, it appears probable that in many sufferers from heart disease the production of emphysema is facilitated in some other way. Basch† has called attention to the fact that cardiac dyspnea due to insufficiency of the left ventricle is followed by descent of the diaphragm and a congestive enlargement of the lungs. He established the general law that any engorgement of the pulmonary vessels produces an enlargement of the lungs, while anything that diminishes the amount of blood in the pulmonary vessels brings about a reduction in the volume of the lungs. This statement is supported by experiments performed by Grossman in Basch's laboratory.‡ An air bladder was introduced into the right heart and distended so that only a small quantity of blood could pass through the pulmonary artery. The experiment was attended by elevation of the diaphragm and a simultaneous reduction in the intrathoracic pressure, so that, according to his reasoning, the lung must have been contracted. When the bladder was introduced in the left heart and distended, so as to offer an obstruction to the passage of blood from the auricle into the aorta, congestion was produced in the lungs, the diaphragm was depressed, and the intrathoracic pressure rose. In this case, therefore, the effectiveness of respiration was diminished. The lung under such circumstances cannot expand as thoroughly as a normal lung; in other words, it has become rigid (rigidity of the lung). In anemia of the lung, on the other hand, as represented by the first experiment, the effectiveness of respiration is increased, for the obstacles to respiration are diminished and the excursions of the anemic lung are greater.

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* Schmidt's "Jahrbücher," 1881.

† Congress für innere Medicin, 1889.

‡ *Zeitschr. für klin. Medicin*, vol. xx. 1892.

It cannot be doubted that the conditions present in cardiac emphysema harmonize with Basch's scheme or theory of pulmonary rigidity, and that in these cases the primary trouble resides in the heart; and while congestive enlargement of the lung has often been classed with ordinary emphysema, it ought, as a matter of fact, to be strictly separated from that condition. But the relations are so complex that the distinction is quite impossible in practice, simple as it may appear in theory. Congestive catarrh of the lung, again, may lead to genuine emphysema—a possibility that has frequently been demonstrated in cases of this kind at the autopsy table. The question of cardiac emphysema is in urgent need of further elucidation. But I must utter a warning against confounding it with the senile emphysema of arteriosclerosis, which is, of course, an entirely different condition, although many transitional forms between the two will no doubt be discovered.

Among **occupations** that are regarded as possible causes of emphysema, playing on wind-instruments has already been mentioned. This occupation obviously tends to interfere with expiration, and accordingly satisfies perfectly the conditions necessary for the production of emphysema which have been erected on theoretic grounds. It was also pointed out in that connection how with the proper precautions the danger incident to this occupation may be minimized. That diseases due to the inhalation of dust are causative principally by the fact that they produce bronchitis is obvious. There are certain conditions that interfere with inspiration much more than with expiration, and these may be, in a sense, antagonistic to emphysema. To this class belong the forms of stenosis that have an opposite effect from that produced by those which lead to emphysema. Most croupous forms of laryngeal stenosis are included in this class. If emphysema develops after diphtheria, it is not simply as a sequel of stenosis of the larynx. Ascites is another condition that is antagonistic to emphysema, inasmuch as it interferes chiefly with the movements of the diaphragm.

Calcification of the costal cartilages appears to affect inspiration more than expiration, the rigid thorax being unable to expand properly. But the powerful diaphragm stands ready to make up for this deficiency. The shape of the thorax, however, is altered by calcification of the cartilages so that it fails to retain its normal position of equilibrium and acquires a constant tendency to assume the inspiratory position; and the rigidity, which prevents it from returning to the normal position of equilibrium, in a still greater degree prevents it from assuming the position of full expiration. The expiratory muscles are then called into action to make up for the deficiency [of expiratory movement], and their inadequacy to do this has already been sufficiently pointed out. Expiration in this condition is therefore impeded more than inspiration, and this view of the question brings out the significance of Freund's observations.

The occasional occurrence of emphysema after pneumonia is now easily comprehensible; the pneumonia is, of course, not the direct cause—that must be sought elsewhere. The disease merely affects the elasticity of the portion of the lung involved, and secondarily that of the entire lung. In this way almost any injury may, under certain circumstances, become causative, and those who use the word "cause" loosely may attribute emphysema to almost anything. But a similar line of reasoning might end in attributing a case of measles to the child's having fallen downstairs. If any scientific advance is to be made, we must avoid the use of such vague phrases.

To find the cause of a disease we must find the factor in the absence of which the disease cannot be produced. In typhoid fever, for example, the presence of the characteristic bacillus is necessary. Typhoid fever need not necessarily exist wherever the bacillus is present; but if the bacillus is absent, typhoid fever cannot be produced. In this sense I consider any interference with the movement of the air in the bronchi, in which the escape of air is hindered more than the entrance, as the cause of emphysema. Clinically the causes of emphysema correspond with those diseases in which an interference with the circulation of air in this sense is present in extensive portions of the bronchial tree. Such diseases, according to my view, are the various forms of catarrh, simple catarrh, asthmatic catarrh, and congestive catarrh; calcification of the costal cartilages leading to rigidity of the thorax; and certain forms of stenosis of the larger air-passages in which expiration is more labored than inspiration. Any other disease, if it produces emphysema at all, does so secondarily. Cantani,* in a discussion of the etiology of emphysema, went so far as to assume a peculiar form of bronchial catarrh, characterized by marked swelling of the mucous membrane extending into the bronchioles and involving the entire bronchial tree. This condition, he says, produces diffuse catarrhal bronchostenosis, which in turn is declared to be the chief cause of emphysema. That emphysema is produced by a condition in which the inspiratory air is able to enter the alveoli with comparative ease, while during expiration the escape of the air is materially retarded, or even in part prevented, by the narrowing of the bronchi, and, in addition, by the occlusion of their lumen with tenacious mucus, is in perfect harmony with the first opinion advanced by Laennec, and I, for my part, entirely agree with that opinion. I do not believe, however, that this condition requires a special form of catarrh; the ordinary bronchial catarrh, providing it lasts long enough, is quite sufficient to bring about the necessary conditions. Cantani still further complicates the question by regarding asthma as a consequence of bronchial stenosis, which is in itself incorrect.

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* Morgagni, 1885, No. 4.

The dyspneic attacks that accompany bronchitic, or laryngeal, or tracheal, or cardiac emphysema are not identical with asthma. To avoid confusion we must insist that asthma is a primary disease resting on a nervous basis, and that the catarrh which accompanies asthma is specific, being characterized by peculiar changes in the sputum, consisting in eosinophile cells, spirals, and crystals that do not belong to ordinary bronchitis.

I am quite willing to admit, however, that Cantani's view is quite correct in one respect. He ascribes quite as much importance to catarrh as I do, only he goes too far in saying that a special form of catarrh is necessary.

The numerous views in regard to the nature of emphysema that have been advanced by other authors appear to me to accord too much scope to the speculative element.

But so many important points of view may be gathered from the study of these theories that I cannot bring myself to overlook them altogether.

For many years the inspiration theory, first advanced in various forms by Laennec and Rokitansky, has stood its ground against the expiration theory, which was first championed by Jenner, Mendelssohn, and Ziemssen. Even at the present day *forced inspiration* as the fundamental cause of emphysema finds its principal supporters in France.

Laennec first taught that excessive inspiration produces laceration, or at least injurious overdistention of the elastic fibers, and thus leads to emphysema. Marfan, who emphatically indorsed this view, supported it by the following experiments and observations:

1. Hirtz* constricted the trachea of a rabbit with a ligature, thus producing dyspnea, and then induced the animal to make excessive inspiratory efforts. The animals in this experiment become emphysematous and die in about a week. If paralysis of the diaphragm is produced by section of the phrenic nerve, the development of emphysema is prevented. This experiment proves nothing—it is so complicated and introduces so many conditions that it is nothing short of audacity to utilize it in favor of the inspiration theory. I consider it merely an argument for the use of anti-vivisectionists. Animals should not be tortured in this way.

2. Asthmatic attacks are frequently the cause of emphysema, but these attacks of dyspnea depend on tetanus of the inspiratory muscles which distend the thorax to its utmost. After each successive attack of asthma the lung remains distended for some time, and finally regains its normal volume. But if the attacks are repeated too frequently, the elastic fibers of the lung finally give way because they are subject to distention for too long a time. The walls of the alveoli, of the infundibuli, and of the acini become perforated and atrophy. The cavities of the lobule coalesce, and permanent emphysema develops. This explanation, which

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* Thèse de Paris, 1878.

begins with a very questionable theory in regard to the nature of asthma, contains not a single convincing factor, and merely illustrates the importance of inspiration; compare this with what follows.

3. Every disease associated with intense dyspnea has the same effect as asthma. The following conditions are then enumerated: croup; stenosis of the larynx, of the trachea, of the bronchi; foreign bodies; capillary bronchitis; bronchopneumonia; pulmonary embolism; mediastinal tumors and other examples of severe grades of dyspnea followed by emphysema. This statement may be utilized quite as readily in favor of expiration as of inspiration, but the force of the argument is destroyed by the fact already touched upon, that numerous cases of this kind occur without producing any emphysema.

4. Experimental emphysema produced by dividing the pneumogastric nerves is said to be an inspiratory emphysema. According to Claude Bernard, the animal is deprived of its pulmonary sensibility, and, being unable to adapt its respiratory efforts to the capacity of the lungs, it inspires with excessive force and so becomes emphysematous. This theory calls for no special discussion, as the authors are not all agreed that animals after section of the vagus die of emphysema; in fact, most of them do not speak of emphysema at all. The theory of emphysema was erected by Longet, and attributed by him to paralysis of the bronchial muscles. There is no doubt that the animals speedily die with the formation of numerous bronchopneumonic foci.*

5. The emphysema occurring in children with adenoid vegetations in the nasopharynx is also explained by Marfan as an inspiratory form, without giving reasons. Compensatory emphysema, which does not belong in this category, is explained by that author in the same way.

It follows from this exposition that I regard all the positive reasons that have been given for the production of emphysema by inspiratory dyspnea alone as extremely doubtful. Marfan's objections to the upholders of the expiration theory are somewhat more plausible. That theory is chiefly based on the fact that cough manifestly favors the development of emphysema, and that the pathogenic element is to be sought in the increase of inspiratory pressure which accompanies the cough. Marfan says that the increased intrapulmonary pressure can act on the elastic fibers only when they have already been overstretched by inspiration. So far he is quite right, but why does he go on to say: "Cough therefore does not act by calling expiratory forces into action; but, like dyspnea, through the agency of inspiratory forces"? There is no doubt that the bad effects of the cough are in direct proportion to the degree of inspiratory distention of the lungs, but it is quite certain that the coughing efforts to which the excessive pressure is due are produced by the contraction of the expiratory muscles.

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* Frey, "Die pathologischen Lungenveränderungen nach Lähmung der Vagi," Leipzig, 1877.

The effect of cough is therefore peculiarly unfavorable because deep inspiration is followed by violent expiration. Bearing down, in which the glottis is closed as for deep inspiration and the expiratory muscles force the diaphragm downward against the contents of the abdomen, has the same effect; but as it is only an occasional and relatively rare occurrence, it does not suffice to bring about a genuine emphysema. In bearing down, also, says Marfan, the expiratory forces are only auxiliary, the principals being the inspiratory forces; although he admits the importance of expiration in this act, he nevertheless arrives at the final conclusion that: "There is only one mechanical cause of emphysema, which is overexertion of the inspiratory forces."

Other arguments adduced by those who represent the expiratory theory are not so utterly worthless as to justify their being passed over in silence. Expiratory pressure may greatly exceed inspiratory negative pressure—in cough as much as 57 mm. of mercury. It may therefore prove correspondingly more dangerous. Those portions of the lungs which are compressed by the thorax and the contracting muscles of respiration are most subject to become emphysematous. During cough the distended lungs are sometimes seen like two sacs projecting above the clavicles. Mendelssohn was the first to point out that the apices of the lungs are more defenseless against increased expiratory pressure than all the other portions of the lungs, because above the clavicles they are covered only by soft parts. During forced expiration, and especially during cough, the lower portion of the thorax contracts because it represents practically the only point of insertion of the expiratory muscles. If forced expiration is made while the glottis is narrowed, the air is forced out of the lower portion of the thorax and part of it enters the upper portions of the lung, where the compression is less. At this point the pressure becomes especially high, and emphysema is therefore most marked in that area. Of course, the positive pressure in the upper portions of the lungs can never be higher than in the lower portions, hence I am not prepared to accept this explanation of the greater tendency to emphysema in the apices. The robust muscular roof of the superior opening of the thorax is not to be despised. In cachectic individuals, however, this circumstance may assume more or less importance.

Emphysema certainly cannot be produced without the cooperation of the inspiratory forces, and as it is quite conceivable that it might be produced without expiration, it would seem as if there was more in favor of inspiration. But as the question is not decided by speculation, but by the observation of natural processes, it must be admitted that in the great majority of cases emphysema is not produced without bronchial catarrh and cough, and that in these cases a certain amount of injury to the alveoli, partly by inspiratory traction, but still more by expiratory pressure, is inevitable. There may be isolated cases in which the emphysema is either exclusively inspiratory or exclusively expiratory. But in view of the difficulty of accurate observation, I fear that such cases frequently owe their existence to one-sided reasoning and defective observation.

Thus, Louis has pointed out that emphysema occurs not only in alveoli supplied by already diseased bronchi, but also in alveoli the supplying bronchi of which are perfectly sound. But I consider it absolutely impossible to demonstrate such an observation at the autopsy. Although the bronchi may look perfectly healthy, they may at the same time have been filled with tenacious mucus which is quite enough to produce the emphysema. Louis is quoted as an authority in many books, especially French works, to show that catarrh cannot have the significance ascribed to it by Laennec. Louis' original treatise, which it is absolutely necessary to know before one is in a position to appreciate his standpoint, I was unfortunately unable to procure. It may be quite true that there are plenty of cases of emphysema without catarrh, at least if the statement is extended to compensatory emphysema. I admit also that there are cases in which the apparently healthy portions of the lung are attacked by emphysema because other portions are incapable of performing their functions, and in such cases it may be justifiable to say that the emphysema has in all probability developed without catarrh. The reader is, however, referred to what I have said under the head of compensatory emphysema. The statement does not in the least apply to the form of the disease which is now under discussion. While it may not be possible to draw a sharp distinction between primary and compensatory emphysema, difficult theoretic questions cannot be solved by an appeal to doubtful or ambiguous cases. An assumption that may be perfectly justifiable for certain typical cases of compensatory emphysema is not at all suitable for typical cases of primary emphysema. Another point that Louis might utilize to support his theory is that in acute emphysema, which occurs during dyspneic attacks, especially in children, the cause, according to many authors, is to be sought exclusively in the excessive inspiratory efforts. I cannot, however, even agree to this argument, and adhere to the view that diffuse emphysema cannot be produced by inspiratory over-exertion alone, if there is no greater resistance to the escape of the air than to its entrance. The possibility that a true overdistention of extensive areas of the elastic pulmonary tissue can be brought about by the inspiratory muscles alone seems to me very far fetched, in view of the relatively limited expansibility of the thorax under the most favorable circumstances, and I certainly cannot accept the statement without objective demonstration. I acknowledge that it is a choice between one opinion and another, and I assume that what others have considered possible is impossible. If the advance of science should show that I am mistaken, it will, I am sure, only be in the few cases in which substantive emphysema develops from acute emphysema. In this connection I attach great importance to the observations of two such distinguished men as Rilliet and Barthez, who took occasion to say in their work on "Pneumonia" (vol. I, p.

76) that "emphysema is usually bilateral, like pneumonia; if the latter is unilateral, the emphysema is usually limited to the affected side. It is always most pronounced where inflammation is greatest." If emphysema were due to the effect of inspiratory overexertion alone, it must be most marked on the sound side, as there can be no doubt that here the inspiration makes itself felt most forcibly. On the affected side it is actually diminished! Evidently the morbid process has a good deal to do with the etiology. Waldenburg tells of a medical student who came from a small town and was not in the habit of climbing stairs; he took lodgings in Berlin on the fifth floor of a house, and became emphysematous by this constant going upstairs. To this may be added another case reported by Hertz*: "A young man twenty-five years of age, in the course of his daily work had to carry pieces of cloth up a very steep stairs a great many (!) times a day. In the course of a year all the subjective and objective signs of emphysema developed, although the man had never in his life suffered from bronchitis or cough." The same author in the same paper mentions a case of Peppers: A man thirty-seven years of age, who was employed in a factory, developed emphysema without having had cough or bronchitis. I do not believe that such cases as these are sufficiently authenticated to form the basis of a theory; even the diagnosis of emphysema appears to me doubtful. Emphysema is so apt to be diagnosed when a certain degree of cardiac weakness and obesity are present. Isolated observations of this kind may be worthy of some attention; but to be utilized to prove anything they would have to be much more numerous and much more accurately observed and reported.

The controversy over the question whether inspiration or expiration is the essential factor in the causation of emphysema naturally directed the attention to the **mechanism of respiration.** The authors who took a prominent part in this controversy were regarded as the representatives of a mechanical theory, and many critics, particularly among the pathologists, pointed out that the changes in the pulmonary tissue had not been sufficiently considered; the importance of the nutritive disturbances was to be placed over against the mechanical theory. For my part, I believe that this criticism went too far in the other direction. The so-called representatives of the mechanical theory were quite aware of the significance of the nutritive disturbances. The most recent investigations in regard to the more minute changes in the emphysematous lung threw a clearer light on the importance of the nutritive disturbances. A group of authors thus arose who might be designated the representatives of the dynamic theory, and who, neglecting to a certain extent the mechanical conditions, regarded the nutritive disturbances as the most important factor. But many even of those who devoted themselves specially to microscopic investigations are found in the ranks of the supporters of the mechanical theory.

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* "Lungenemphysem," p. 452.

Some others have even gone so far as to say that there is a specific disease of the pulmonary tissue, possessing a special tendency to the formation of holes in the interalveolar septa, with ultimate disappearance of these structures. This is a purely speculative theory. The disease has never been demonstrated by observation, and its existence rests solely on postmortem findings. The objections raised by Virchow* against the production of emphysema by obstruction do not affect the mechanical theory, because no one pretends to say that emphysema is produced by actual occlusion. His reasons for the distribution of the pigment prove nothing, because the pigment is not firmly attached to the tissue and can be removed even in old age. But his theory is so carefully formulated that it is accepted by many, even at the present day, and as it is that of a prominent authority, it deserves to be quoted here: "I am much inclined to assume the existence of a kind of primary disease in the pulmonary tissue, a disease the predisposition to which is developed in early life, and which becomes gradually more marked as time goes on. On the other hand, however, I once more wish to emphasize that I am far from asserting that the pressure of the air contained in these alveoli has nothing to do with the further development of this condition; on the contrary, this pressure is in my opinion absolutely necessary. . . . But whether the defective expiration is always due to mechanical obstructions in the respiratory passages is another question. It is evident that if, for example, the pulmonary tissue suffers a material loss of its elastic properties,—and this has been demonstrated to take place after inflammation,—the retention of air in the alveoli and the maintenance of the respiratory conditions become explicable." I am willing to admit all this, but the assumption of a primary disease I regard as absolutely theoretic and altogether superfluous. It is another thing to concede that the lungs of different individuals possess different powers of resistance, and that many persons, owing to their peculiar constitution, are especially apt to acquire emphysema. Such a constitutional weakness may be acquired or congenital. It is to be regretted that the characteristic signs of this condition are as yet unknown. On the other hand, I do not believe myself justified in discarding altogether the influence of heredity, although it requires to be much better supported (see the cases cited on p. 264). Every busy practitioner has observed how emphysema sometimes becomes especially prominent in a certain family, and no one doubts that hereditary factors have a certain influence. While it may be quite possible that now and then an individual may develop emphysema without any other demonstrable cause than hereditary predisposition, it is not enough to assert that such a thing is possible. Convincing cases must be collected, and there lies the difficulty.

We have the statements of Jackson and Greenhow † in regard to the influence of heredity. The former states that one or the other of the parents was affected in 18 among 28 emphysematous subjects; and, according to the latter, 23 among 42 cases gave a positive history of hereditary predisposition to the disease (see also under Etiology). Lebert,* on the other hand, analyzed 100 cases, and in only 13 of these had reason to believe that pulmonary emphysema existed in the parents. Greenhow regards a gouty diathesis as especially predisposing to emphysema, because he was able to demonstrate gouty symptoms in 12 of his patients or their near relatives. Norman Moore† also believes that emphysema is especially apt to develop in gouty subjects. In justice to these authors, the theory that a gouty constitution is associated with a special predisposition to emphysema must at least be taken into consideration. For my part, I believe that so-called gouty emphysematous subjects are really alcoholics. Lebert also recognizes a constitutional emphysema. He asserts that in at least 7% of his cases emphysema was demonstrable in early youth without deformity of the thorax and without pulmonary catarrh. I am still convinced that these statements rest on inadequate observation.

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* *Berliner klin. Wochenschr.*, 1888, No. 1.

† *Lancet*, 1867, II.

The opinion of such a distinguished and able investigator as Villemin cannot be disregarded. He devoted an extensive article‡ to the subject of emphysema. According to him, the walls of the alveoli contain nuclei which hypertrophy and effect compression of the capillaries. As a result, secondary nutritive disturbances take place in the alveolar walls and in the nuclei themselves, which undergo fatty degeneration and are converted into sputum. In this way the alveolar walls disappear and several alveoli coalesce. Interlobar and subpleural emphysema is also produced in this way. The elasticity of the alveoli is impaired by this disease and they retain a gradually increasing quantity of air. The cough, which even in the first stage of the development of emphysema is short and dry, is produced reflexly, according to Virchow, by the morbid process in the alveoli. The bronchitis, on the other hand, is secondary; and this, he says, explains the observations of Louis, Andral, and Hervieux of emphysema without bronchitis or antecedent to bronchitis. Cough causes laceration of the altered alveoli, and thus hastens the development of an emphysema; the influence of the cough is then reinforced by the bronchitis. According to his view, therefore, the primary cause is a tissue change due to some hitherto unknown cause, a view which, like that of Virchow quoted above, rests on insufficient observation. The cases without bronchitis or preceding bronchitis are open to criticism, and do not appear to me for the present sufficiently authentic to serve as a basis for a theory.

Freund § finds the cause of emphysema in a primary nutritive disturbance of the costal cartilages; he gives a very thorough, though unfortunately somewhat unintelligible, explanation of the mechanism of the ribs, and the importance of this mechanism is recognized by every one who has written on respiratory movements. If this mechanism is injured, the respiration must suffer—of that there is no question. That the mechanism becomes greatly damaged in emphysema is also above dispute, and Freund deserves credit for bringing convincing proof of this fact. He began by studying the texture of the costal cartilages, the changes in which he described very accurately. An increase in the size of the cartilage is brought about by these changes and, as it is placed between two bones,—the sternum and the rib,—it constantly tends to force these bones apart. As the process gradually advances, the rib is forced after a long time to make an inspiratory movement. In the same way the sternum is forced forward and upward. "The result of this anomaly is, in the first place, that the constant expiratory space of the thoracic cage gradually expands in all directions, beginning with the anterior circumference, and, in the second place, that inspiration, which in part is *preoccupied* (!) [ushered in?—Ed.] in the inspiratory position, becomes less effective, as does also the expiration, because the latter is unable properly to empty the air-distended lung. The cause of pulmonary emphysema is to be found in these changes of the shape and function of the thoracic wall, instead of these changes, as has hitherto been thought, being the consequences of emphysema."

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* Klinik der Brustkrankheiten.

† *Path. Soc. Trans.*, vol. XXXIII.

‡ *Archives générales*, 1866, II, p. 566.

§ "Der Zusammenhang gewisser Lungenkrankheiten mit primären Rippenknorpel-Anomalien." Erlanger, 1859.

The results of Freund's investigations afford a good illustration of the difficulty of distinguishing cause from effect in medicine. The alterations are so involved and the relations so complex that it is quite impossible to say, when the disease has become well developed, what is primary and what is not. Such questions cannot be decided by ratiocination.* The interpretation of the facts observed in the formative stage of the disease is exceedingly difficult, and often leaves us in the lurch. For the present I believe that, while certain diseases of the air-passages are to be regarded as the cause of emphysema, there are also cases in which the rigidity of the walls of the thorax manifests itself as the primary lesion, and this I believe to be especially true in cases of senile emphysema and emphysema of arteriosclerotic individuals and persons suffering from certain forms of heart disease. But Freund is mistaken in thinking that the lung is absolutely powerless to resist the action of the thoracic wall, and if he cites the authority of no less a person than Donders in support of his theory, the only answer is that Donders also was mistaken. I quote Donders' instructive passage *in extenso*. He is speaking of the diaphragm: "It has been said that the distended lungs force the diaphragm downward; but whence do they derive the power that enables them to do this? Is it not true that the shape of the lungs, as long as they are distended, is determined by the shape of the thoracic cage? and, if so, how can the lung, which in emphysema is excessively distended, modify the shape of the thoracic cage?" Donders overlooks the fact that when the lung is distended with air, and the air is unable to escape, the diaphragm is prevented from returning to its normal position of relaxation. The lungs do not exert active compression, but they do offer resistance, which is quite enough. And just as the lungs are able to influence the diaphragm, so they are also able to influence the shape of the thorax.

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* A "law" such as that formulated by Freund is quite properly rejected by Fournet.

It would be as one-sided to say that the shape of the thorax is determined solely by the lungs as to say that the shape of the lungs can only be determined by the thoracic wall. In each case morbid processes in the one react on the other by producing symptoms. The one-sided theory is therefore quite as erroneous as the one-sided theory in regard to the lungs. Observation shows, so far as we are able to judge of the question now, that in the majority of cases the pulmonary theory holds good, but that there is also a good deal of room for the thoracic theory. J. Lange * advanced the theory that emphysema is produced by a paralytic neurosis of one or several of the nerve-branches distributed to the finest bronchioles and innervating the bronchial muscles. The contractile power of the lung is diminished; as a result the residual air is increased, and the elastic fibers are stretched beyond the limits of elasticity. It is not inconceivable that the advance in neuropathology might put us in possession of cases of emphysema originating according to Lange's theory, but for the present it must still be regarded as purely speculative. I refer to Ziertmann's communication cited above.

Grawitz,† in connection with his views in regard to connective tissue and connective-tissue cells, also advanced a theory about emphysema. Criticism of these views is out of the question in this volume; that may safely be left to the pathologists. Grawitz refuses to have anything to do with mechanical influences; he is one of the most extreme supporters of the dynamic theory. He discards the mechanical theory on the ground that the portions of the lung behind an obstruction in the bronchi become atelectatic ‡ and the forced expulsive movements during cough produce an interstitial and not a genuine emphysema. He then calls attention to the fact that emphysema occurs especially in the anterior, sharp border of the lungs, and that the emphysematous areas contain very little coal-dust. From this he concludes that the causative element is not derived from the air-passages, but from the circulation. He distinguishes a dropsical emphysema, which develops chiefly in persons suffering from heart disease, an inflammatory, and a senile form. The slumbering cells in the ground-substance are awakened into action by the primary injury; while before they were invisible and refused to take a stain,

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* "Ueber das substantive Lungenemphysem und dessen Behandlung," Dresden, 1870.

† *Deutsche med. Wochenschr.*, 1892, No. 10.

‡ He evidently agrees with Virchow. I must insist, however, that the bronchi are not assumed to be occluded, but merely obstructed so as to interfere with the free circulation of air.

they now make their appearance and become active, take up particles of coal-dust with which they enter the lymph channels and are carried off. The injury that is responsible for the development of this process and leads to the disappearance of the alveolar septa and of the connective tissue in the dropsical form is a disturbance of circulation; in the inflammatory form, an inflammatory irritation which is not specific; in the senile form, deficient nutrition owing to the decay of the circulatory apparatus. The same applies to the elastic fibers. They also form tingible nuclei at definite intervals. The adjoining segment of fiber gradually becomes granular and the elastic fibers are thus converted into spindle cells.

According to Klaesi, the excessive dilatation of the alveoli is the primary cause of the detachment of the epithelial cells from their foundation and from one another, the detachment being most active in the capillaries. This leads secondarily to an impairment of the nutrition and a reduction in the quantity of water (?) contained in the walls of the capillaries. The epithelial cells degenerate, leaving only a thin lamella incapable of offering any resistance, especially in the alveolar septa, where the degenerative process first makes its appearance. This lamella disappears by absorption or degeneration, and the elastic fibers embedded within it also disappear or retract and surround the opening. The larger this opening, the thicker *ceteris paribus* is the layer of elastic tissue, the formation of which is explained by a gradual pushing apart of the fibers. The epithelial degeneration ushers in the disappearance of the capillaries; the capillaries have lost their support. By distention and dilatation their lumen is reduced to a minimum, the walls are brought into apposition, degenerate, and finally disappear.

In this explanation the increased pressure in the alveoli is really regarded as the origin of the disturbance, and Klaesi's theory is not improperly characterized as a purely dynamic theory.

Auld * also reached the conclusion that the changes in the capillaries are always preceded by alterations of the epithelium and of the connective tissue.

The theories of Thierfelder,† Rindfleisch,‡ Hertz, Eppinger,§ and Rainey,‖ based on accurate observations of the microscopic conditions, practically admit the existence of some form of injury which brings about distention and increased pressure in these alveoli, followed by alterations of the texture of those structures. Jackson insists particularly on the degeneration of the capillaries; Klob ** speaks of cell proliferation in the capillaries with resulting obliteration.

Bayer †† made a special study of the nuclei said by Wagner to be present in abundance in the tissues of emphysematous lungs. They are evidently the same as those observed by Villemin, which Biermer regards as the nuclei of epithelial cells. Bayer, however, found that, in addition to these, other nuclei are undoubtedly found in the connective tissue, which must be regarded as true stroma-nuclei independent of epithelial cells, capillaries, or smooth muscle-fibers. He acknowledges that the multiplication of the structures that he observed is only apparent, being simulated by the fact that the stretched walls, due to the emphysematous distention of the alveoli, simply bring a greater number of nuclei into the same place. He believes, however, that in certain old cases of emphysema a true increase in size and number of the nuclear elements takes place, and that in the cavities originally produced by atrophy a secondary thickening of the walls occurs. Accordingly, he refutes the theory of Villemin and assumes a modification of the circulatory conditions—reinforced by mechanical agencies—as the basis for development of vesicular emphysema, excepting only those cases that develop rapidly under the influence of special forces. This modification of the circulation may, of course, be brought about in a variety of ways. Bayer even mentions the possibility of a primary loss of elasticity in the pulmonary artery. He acknowledges that there are a great many possibilities, and freely admits the importance of violent causes of a purely mechanical nature.

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* *Lancet*, 1893.

† "Atlas," 1872.

‡ "Lehrbuch der pathologischen Gewebelehre."

§ *Prager Vierteljahrschrift*, 1876, vol. cxxxii.

‖ *Brit. Med. Jour.*, June 26, 1868.

** Oppolzer, "Vorlesungen uber specielle Pathologie und Therapie."

†† *Archiv der Heilkunde*, xi, 1870.

According to Caprozzi, Morgagni as early as 1870 stated that emphysema is a result of certain forms of bronchitis which render the alveolar walls edematous and thus destroy their elasticity. I regret that I was not able to obtain the original paper. Although pathologic investigations have done much toward clearing up our ideas on this question, it cannot be said to have been demonstrated that the primary cause of emphysema resides in a diseased condition of any one tissue. The importance of the mechanism of respiration asserts itself everywhere, and in the great majority of cases, after a minute scrutiny of the tissues of the lungs, one is forced to take a stand in favor of the mechanical theory.

Changes in the mechanism of respiration in healthy individuals not exceeding the limits of health can never in themselves lead to emphysema; so much is clear *à priori*, and has been established again and again by numerous discussions and observations, among which may be mentioned the observations made on singers by Lagrange and Wassiljew and on mountain climbers by Fortassini. There must be some actual pathologic change which cannot remain purely mechanical. Even though the original injury is mechanical, dynamic effects are at once produced, and if the injury is dynamic to begin with, the mechanical features at once contribute their deleterious influence. For these considerations it will be difficult to find any one at the present time willing to take an exclusive stand. There is an indissoluble connection between disturbances of mechanism and impairment of nutrition.

To continue the search after the primary cause is a mistake, simply because clinical observations have shown that, so far as is known, there is no primary cause for emphysema. Emphysema is not a primary disease, but a sequel of various diseases, the terminal stage of various diseases which injure the mechanism of respiration in similar ways and produce similar nutritive disturbances in the pulmonary tissue.

We are forced, therefore, to assume an intermediate position, and this has now been done by most authors. Biermer* first laid the foundations for this intermediate theory, Niemeyer and Gerhardt accepted it; the works of Hertz, Homolle, Koranyi, and Liebermeister, and, more recently, the writings of Strümpell, Fleischer, and Lenhartz, give expression to a similar view. These authorities, unlike former writers, abstain from setting up a theory in regard to the nature of the disease and from trying to force an explanation of everything that does not at once fit in with such a theory.

It is worth noting that certain diseases have been regarded as antagonistic to emphysema. No less an authority than Rokitansky says that *emphysema and pulmonary tuberculosis occupy a relation of mutual exclusion.*† As a matter of fact, emphysema is found often enough in tuberculosis; but it is a compensatory emphysema, and does not belong to the present discussion. On the other hand, an emphysematous lung is a distinctly unfavorable soil for the development of tuberculosis. This is explained in various ways. Some believe that the atrophic anemic condition of the lung is a reason why the tubercle bacilli fail to find favorable conditions for their propagation; others that the insufficient inspiration is unfavorable for the transportation of the bacilli. For my part, I am not inclined to attach weight to this relation of mutual exclusion. It is a matter of observation both that tuberculous subjects may become emphysematous, and that emphysematous subjects may develop tuberculosis; only one disease masks the symptoms of the other or even renders the diagnosis impossible. There is a want of well-sifted and well-observed cases bearing on the question, and a statement based on general impressions is very apt to be misleading.

Even Louis and Fauvel cite cases of miliary tuberculosis with emphysema. Burkhardt observed miliary tuberculosis in 12 out of 18 cases of emphysema, and in recent times Heidler ‡ observed another case of tuberculosis in an emphysematous subject. While I am engaged on this manuscript, there is brought to me a section of a lung which on the left side is phthisical and adherent to the thoracic wall by a thick plastic exudate, while the right side is typically emphysematous. Both lungs are uniformly covered with innumerable miliary nodules.

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* "Handbuch," edited by Virchow.

† Koranyi distinctly says that emphysematous subjects comparatively rarely develop pneumonia, and still more rarely tuberculosis.

‡ *Wiener med. Wochenschr.*, 1889, Nos. 4, 5.

Bouillaud, Oppolzer, Biermer, and others believe that there is a relation of mutual exclusion between *emphysema and cardiac disease.* They say that emphysematous subjects are less disposed to develop rheumatism and cardiac lesions. Biermer adds that, although slight grades of emphysema are sometimes observed in conjunction with valvular lesions, the emphysema in such cases is to be regarded chiefly as a consequence of the respiratory disturbances due to the cardiac lesion. In answer to this, it may be observed that emphysema in its most classic and most familiar form is nothing more than the consequence of respiratory disturbances dependent on bronchial catarrh.

Certain views have been advanced in connection with the answer to the question whether mitral or valvular lesions are apt to be associated with emphysema. Marfan says that emphysema frequently accompanies aortic disease. Biermer is of the opinion that atheroma is a more frequent accompaniment of emphysema than endocarditis, and it is admitted that atheroma is the chief source of aortic disease. On the other hand, the diseases of the myocardium have been unduly neglected, for they undoubtedly predispose to pulmonary catarrh and emphysema. There is no question in my mind that many conditions were formerly diagnosed as emphysema which on more careful examination would have been regarded as myocarditis. The trouble in the past has been that emphysema was too much regarded as an idiopathic, primary disease. It is necessary to become familiar with the thought that it is not a primary disease, but only a sequel.

Biermer concludes his remarks on the subject of mutual exclusion by saying that emphysematous patients appear, on the whole, to be less disposed to croupous pneumonia and other acute inflammatory diseases. There is no doubt that hepatized emphysema is rarely seen at the autopsy table.

COURSE.

Diffuse emphysema occurs in various forms. An acute form might first be distinguished. Any excessive respiratory exertion in acute diseases, in which there is some obstruction in the air-passages, may in a short time lead to the development of a high grade of distention in the lungs. The distention, as a rule, only lasts a short time, and disappears along with the subsidence of the primary disease.

In such cases the term "*acute pulmonary distention*" instead of "emphysema" is usually employed; for, it is said, emphysema implies the existence of characteristic pathologic changes, such as atrophy of the walls, dilatation of the pore-canals, disappearance of the capillaries, for the production of which no time is afforded in acute pulmonary distention.

It is, of course, impossible to draw a sharp distinction between pulmonary distention and emphysema. From a pathologic standpoint it may be said that emphysema is present only when the above-described changes are present in the epithelium and in the blood-vessels of the lungs. In practice we speak of pulmonary distention when the condition occurs rather suddenly in the course of an acute disease and there is reason to hope that the lung is still capable of perfect regeneration, although the physician is always prepared for the transition to genuine chronic emphysema. Thus, when a foreign body, for example, has entered the lung, an obstinate cough results which may lead to pulmonary distention. Koranyi relates the case of a child with a melon-seed in the right bronchus. Violent cough and attacks of asphyxia supervened, and within a month distention, chiefly on the right side, but also noticeable on the left, could be demonstrated. Four months later a fortunate accident brought about the expulsion of the melon-seed, which had meanwhile become coated with a fine layer of calcium, and within a month of the expulsion of the foreign body the lung had returned to its normal boundaries. This case can, of course, be regarded as one of acute emphysema, or it may be designated as a case of pulmonary distention of unusual duration, not deserving the name of emphysema. Since every asthmatic attack is unquestionably accompanied by pulmonary distention which gradually becomes constant, why should we hesitate to speak of emphysema in asthma? Where does distention end and emphysema begin? Sharp distinctions always are and always will be unsatisfactory; they are purely artificial; and nature, in the wealth and variety of cases, laughs at classification. It is, however, demanded in a work of scientific description, and we therefore speak of *emphysema in all cases in which pulmonary distention has existed longer than a week.* Some authors decline to call such cases emphysema if they later undergo involution, and suggest the term permanent inspiratory alveolar ectasis. For my part, I cannot see that anything is gained by the introduction of this new term except increased confusion. We call emphysema acute when the condition ends in a return to the normal, and we call it chronic when it remains permanent. This distinction is all the easier as emphysema of one year's duration is certain not to undergo involution and becomes chronic beyond the possibility of a doubt. The question whether emphysema is destined to be acute or chronic could easily be determined beforehand by a consideration of the mode of origin and the age of the patient.

Acute emphysema usually belongs to early youth. After a certain age, usually as early as the second decade, the elastic fiber has so far lost its youthful characteristics that it is unable to return to its normal condition after being overdistended.

Acute emphysema begins in the course of any acute disease that is attended with marked interference with the free circulation of air in the bronchial tree. Diseases characterized by violent cough, and therefore by sudden marked increase in the pressure within the bronchial tree, offer special opportunities for the production of pulmonary distention and acute emphysema, since any distention of the lungs may form the starting-point for emphysema. The condition develops most frequently after whooping-cough. A few weeks after the beginning of whooping-cough emphysema may be completely developed. In favorable cases recovery is possible, but in unfavorable cases asthmatic attacks make their appearance and chronic emphysema finally remains.

The same is true of croup and measles, and to a much less degree of pneumonia, although cases of emphysema are not unheard of in the latter disease. Individuals who imprudently exert themselves after pneumonia or other infectious diseases often develop emphysema. Two cases might be cited of enthusiastic wheelmen, one of whom after pneumonia, and the other after typhoid, contracted catarrh as a result of overindulgence in his favorite pastime, and later developed an emphysema. I have no doubt that any infectious disease may become a menace in this way.

True primary emphysema may, therefore, develop from acute pulmonary distention, but the great majority of cases are chronic from the outset and begin with bronchial catarrh. The latter may be a simple catarrh without any complications whatever. If it persists long enough and the secretion remains tenacious and difficult to expectorate, as is the more likely to happen in chronic catarrh the longer the disease lasts, emphysema is the inevitable result. In such a case emphysema is not a special disease at all; it is simply the second stage of chronic bronchitis. Exactly the same thing is true of the numerous varieties of emphysema that follow asthma. Asthma is even more certain than simple catarrh to produce emphysema, for the peculiar tenaciousness of asthmatic sputum is the etiologic factor *par excellence.* Here also it may be said that emphysema is not a disease *per se;* by its development the picture of asthma is simply modified, and emphysema becomes a second stage in the clinical picture of asthma. When the other forms of emphysema are examined, the same peculiarity is observed. In alcoholics, in heart patients, in arteriosclerotics, emphysema is always preceded by some other morbid condition which gradually leads to the development of the disease. The only exception is to be found in performers on wind-instruments who do not possess the skill or the powers of resistance requisite for their calling. In these, emphysema may sometimes appear as a primary disease from the outset. But even in these cases it is not probable that the condition is quite so simple.

At any rate, when the emphysema has attained a certain degree, it so dominates the clinical picture that it may be regarded clinically as an independent disease; it gives rise to a very definite symptom-complex and leads to very characteristic consequences no matter what the manner of its origin may be. Numerous variations in the severity of the symptoms and in the order in which they appear, and variations in the appearance of the sequels, nevertheless depend on differences in the beginning of the disease. Bronchitic emphysema is not exactly like asthmatic, and certainly not like arteriosclerotic emphysema or the form observed among glass-blowers, although the main features may coincide. But the general agreement is so marked; the various secondary forms present such numerous minute differences in symptomatology and in the clinical course; the transition from one form to another is so gradual, that the attempt to set up a further subdivision has so far failed to secure general approbation. I shall return to this point at the end of the article.

As soon as a chronic emphysematous condition has developed, the patients become dyspneic and are constantly troubled by a liability to catarrh. Their comfort is very much dependent on the weather; during the inclement season they feel worse, and during the summer they feel better. A certain degree of anemia develops. They gradually become less and less able to work; soon a very short walk becomes a great effort. The dyspnea periodically becomes aggravated and violent attacks are observed which closely simulate asthmatic attacks; it is found, however, that only those who are true asthmatics had suffered from these attacks before they developed emphysema; in all other patients they occur for the first time after the emphysema has developed. In many cases it is quite impossible to distinguish asthmatic from pseudo-asthmatic attacks.

The subsequent course of the disease depends very much on external circumstances, and on the kind of life the patient leads. Working people as a class suffer most from the disease, partly because they cannot take proper care of themselves, and partly because they injure themselves by their bad habits and reckless way of living. The rich man, however, if he is imprudent, is no better off. The heart becomes involved and the patient enters on a precipitous path which sooner or later carries him down hill to the end. Even in this stage an astonishing degree of improvement is often attainable, but it is rarely very lasting. The relations are extremely complex. The compensatory hypertrophy of the right ventricle may be impaired by malnutrition, and fatty degeneration of the heart, although it is still to a certain extent amenable to treatment; if, however, atheroma of the coronary vessels develops, with the formation of foci and wheals, regeneration is impossible. Even simple degeneration of the heart muscles ultimately becomes irreparable, stasis becomes chronic, and the patients appear either as miserable, cachectic, pale individuals with pronounced edema, dropsy, and anasarca; or strong-looking men with bloated features, and blue discoloration of the mucous membranes and prominent portions of the body—the condition known as akrocyanosis, in which the nose, chin, fingers, toes, knees, and elbows are especially discolored. Even in this more robust class of patients edema makes its appearance, and the enlargement of the liver and albuminuria that are present indicate considerable accumulation of slowly circulating blood in the large glands of the abdomen. There are, therefore, anemic and cyanotic emphysematous patients. Of course, there is no lack of transitional forms between these two extremes, but for practical purposes I prefer to keep the two separate.

The cyanotic patients are, as a rule, those who have suffered from bronchitis, asthma, or heart disease, and those who are alcoholic; the anemic group includes old people and arteriosclerotic persons in general. In the former group dyspnea is usually more intense and the signs of stasis are more conspicuous. In the latter, the patients, who do not complain so much of dyspnea, are atrophic, physically depraved individuals. The chest, instead of being enlarged, appears smaller than normal. The intercostal spaces are unusually wide; the ribs are displaced downward, the lowest pair being found near the crest of the ilium. The cardiac dulness is not masked by the pulmonary resonance and the position of the diaphragm is not lower than normal. It is true that the heart becomes atrophic; but, as the whole body is also atrophic, the heart suffices to distribute the blood required, there is little tendency to hypertrophy of the right ventricle, and the symptoms of stasis may be altogether wanting. In addition to these cases, however, which bear the stamp of senility, there are others, represented usually by vigorous individuals, and characterized by the development of a stage in which the cardiac phenomena become so prominent as completely to overshadow the emphysema. The patient, although his malady began as a simple emphysema, has become a heart patient. The heart muscle degenerates, the symptoms of stasis increase, and the patient succumbs with symptoms of cardiac insufficiency, enlargement of the liver, albuminuria, swelling of the legs, and effusions in all the cavities of the body. Not infrequently some intercurrent disease terminates the patient's life. Rarely an emphysematous patient dies suddenly in an asthmatic attack. In these cases the attack is not one of true asthma or of emphysematous asthma, but rather the genuine cardiac form.

For didactic purposes the course of emphysema in the great majority of cases may be divided into two stages: the catarrhal stage and the stage of cardiac insufficiency. A division into three stages has also been made: catarrhal, cyanotic, and asystolic. It is a characteristic feature of the disease that the symptoms gradually transfer themselves from the lung to the heart.

Even the type of dyspnea ultimately undergoes a change; the difference between inspiration and expiration, which is so marked in emphysema, becomes obliterated and cardiac dyspnea develops. The number of respirations is increased, and inspiration becomes quite as laborious and prolonged as expiration. The asthmatic attacks which occur in this stage are attacks of cardiac asthma—that is to say, attacks such as may occur in any form of cardiac insufficiency; they cannot, however, be sharply distinguished from the dyspneic attacks of emphysematous patients.

If I may be permitted to select certain types from the variegated and complex picture of this disease, for the purpose of explaining its nature, I should propose the following: The first type is *bronchitic emphysema,* where the trouble begins in a simple bronchial catarrh; this form usually occurs in drinkers and workers in dust. The next important type is *asthmatic emphysema,* the accompanying catarrh in which is distinguished by the peculiar sputum; typical asthmatic attacks occur from the beginning and nervous elements peculiar to asthma are obtained from the history. My third type is *cardiac emphysema,* in which the first beginnings can be traced to cardiac phenomena, the presence of valvular lesions, of myocarditis, and of pericardial adhesions usually due to some acute disease, possibly also to syphilis; and in which emphysema quite frequently never attains the dignity of an independent disease. Finally, there is senile emphysema, a group that also includes arteriosclerotics and those individuals who for other reasons have prematurely calcified costal cartilages. In this type the symptoms of catarrh are ill defined or altogether absent.

In practice, acute emphysema, developing after whooping-cough or other infectious diseases, from the aspiration of foreign bodies, and from stenosis of sudden production, may, if desired, be distinguished as a special form. If it does not subside, or if it undergoes some special modification as the result of the causal factor, but maintains the character of emphysema, it will become converted, according to circumstances, into a bronchitic, an asthmatic, or a cardiac form.

A number of cases will, however, be found that cannot be included in any one of these groups. The emphysema is often overshadowed by other important disturbances,—such, for example, as stenois of the air-passages,—and the existence of diffuse emphysema is only discovered accidentally or at the autopsy.

Bronchitic and asthmatic emphysema have been grouped together as hypertrophic forms in contradistinction to senile or atrophic emphysema. The term hypertrophic is, however, condemned by Jenner * as unsuitable in emphysema, and with good reason. The lung is enlarged, but what is it that hypertrophies? He prefers to distinguish a large-lung and a small-lung emphysema. Cardiac emphysema also belongs to the large-lung variety (see page 263). His classification failed to secure general acceptance. If the standpoint is adopted that emphysema is not a disease in itself, but only represents a stage in the course of other diseases, a sufficiently clear theoretic notion of the condition will be obtained. In practice the present custom is destined to remain, since it is so convenient and so simple to group together a large series of symptoms in one clinical picture, even though they may be etiologically quite distinct.

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* Reynolds, "System of Medicine."

SYMPTOMATOLOGY.

(1) *Methods of observing respiration:* (a) *spirometry;* (b) *pneumatometry;* (c) *respiratory curves;* (d) *diaphragm;* (e) *remaining inspiratory muscles;* (f) *expiratory muscles;* (g) *absorption of oxygen and elimination of carbon dioxid.* (2) *Dyspneic attacks.* (3) *Enlargement of the lungs.* (4) *Shape of the thorax.* (5) *Effect on the vertebral column.* (6) *Effect on the sternum.* (7) *The emphysematous habit.* (8) *Palpation.* (9) *Percussion.* (10) *Mensuration.* (11) *Auscultation.* (12) *Catarrh.* (13) *Heart.* (14) *Circulation.* (15) *Liver.* (16) *Spleen.* (17) *Stomach and Intestine.* (18) *Urinary apparatus.* (19) *Genital apparatus.* (20) *Larynx and upper air-passages.* (21) *Nervous system.* (22) *Extremities.* (23) *General nutrition.*

1. **Dyspnea** is considered as the most conspicuous, the most constant, symptom of pulmonary emphysema. A large number of authors have devoted their time to the study of respiration. As a rule, the breathing is somewhat accelerated, inspiration shorter and more hurried, expiration in proportion considerably prolonged. The number of respirations is usually increased, but this is not necessarily the case; in mild grades of the disease it is often normal, but as the disease progresses the number gradually increases, although it never becomes excessive. If the respirations are from 28 to 32 when the patient is at rest, it is a sign that the disease is in an advanced stage or that there exists a temporary exacerbation, especially as the result of catarrh. The respiratory movements are always labored, as they are able to bring about only a relatively small degree of expansion. During inspiration the thorax as a whole is elevated without expanding properly. The supraclavicular fossæ, the suprasternal fossa, and the intercostal spaces opposite the attachments of the diaphragm are retracted; during expiration the thorax becomes depressed, and if the patient exerts a moderate degree of abdominal pressure the abdomen becomes hard and board-like and retracts. The apices of the lungs are somewhat protruded in the form of cushions; and it would appear from Friedrich's observation that the lung may bulge in other areas as well if the muscles do not prevent it.

Some time ago, clinical observation of the respiration being deemed insufficient, instruments came into use with which the amplitude of the respiratory movements and the pressure of inspiration and expiration could be accurately determined. The movements of various points on the thorax are recorded for the purpose of obtaining data from which to deduce the duration and depth of the respiration. The simplest, most useful, and most commonly used of these instruments is the spirometer employed by Hutchinson and Wintrich* in their pioneer investigations. The spirometer shows that the capacity of the lung in emphysema is considerably diminished; often as much as

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* See article on "Diseases of the Respiratory Organs" in "Handbuch der Speciellen Pathologie and Therapie," edited by Virchow. Also Fried, Arnold, "Ueber die Athmungsgrösse des Menschen," Heidelberg, 1855.

50%. Wintrich gives a reduction of 20% to 60% of the normal, except in asthmatic conditions. The quantity of residual air becomes progressively increased, but as there is no trustworthy and convenient method of determining its amount, it is usually disregarded in practice. In the healthy subject the proportion between residual air and vital capacity is as 1: 4 or 1: 5. The method of taking these measurements is described in detail by Bernstein.[*]

In addition to spirometry we have pneumatometry.[†] In healthy subjects the pneumatometer indicates an average inspiratory pressure of 70 to 80, and an expiratory pressure of 110 to 130. In emphysema inspiratory pressure is but little increased, or even normal, while expiratory pressure is reduced first to 100 and later to 70. The inspiratory muscles in emphysematous subjects contract vigorously, but the expiratory muscles are unable to make up for the deficiency due to the diminished elasticity of the lungs and thoracic walls, although the muscles are certainly not atrophic. Waldenburg finds expiration deficient in recent bronchitis before emphysema has developed. In emphysema the various degrees of the disease can, according to Waldenburg's statements, be recognized with this instrument. In the mildest grade, in which dyspnea makes itself felt only on exertion and no change can be determined on percussion, expiratory pressure is a little less than inspiratory pressure, instead of being, as in the normal state, materially higher than inspiratory pressure. In severer grades of the disease, when dyspnea is brought on by moderate exercise, and distinct signs of pulmonary distention are elicited on percussion, the difference between expiratory and inspiratory pressure is quite marked, expiratory pressure being reduced to one-half or one-third of the inspiratory force, while inspiratory pressure remains normal or even becomes greater than normal to compensate for the diminished expiratory pressure. In the higher grades, finally, which accompany bronchitis of many years' standing, with great dyspnea and asthma, cyanosis, edema, and rigidity of the thorax, both the expiratory and the inspiratory pressure is diminished, although the latter is always somewhat greater than the former.

It may seem astonishing that such simple diagnostic data are scarcely, if ever, utilized in practice, and that even in the clinic the pneumatometer has entirely gone out of use. But any one who has worked with the instrument knows that the sources of error are numerous and the conditions in most cases of emphysema so complex that the formula above referred to has merely an academic value. Waldenburg ascribed an altogether extravagant value to these observations.

Biedert[‡] and Krause[§] freed the method from its coarser fallacies and attempted to make it more trustworthy and more profitable. But in spite of their efforts it is not as yet perfect enough to be of any practical use, nor is the scientific significance to be valued too highly. I believe, however, that by careful improvement a high degree of efficiency may in the end be attained. Basch recently attacked the question from another side, which appears to promise better success. At the Seventeenth Congress for Clinical Medicine he exhibited a very interesting apparatus * which he calls a pneumometer. It is intended for measuring the volume and elasticity of the lungs. A detailed description and discussion of the rationale of its use will be found in an article by Pflüger.† To what extent it will prove practically useful I do not pretend to foretell at the present time. I believe, however, that satisfactory results will be obtained only with very intelligent patients and in the hands of expert observers. A method that is capable of determining the elasticity of the lungs numerically cannot fail to give the most valuable data for diagnosis, as well as for differential diagnosis, in emphysema, and may also help to clear up our ideas on the nature of the disease.

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[*] *Pflüger's Archiv*, vol. L.

[†] Waldenburg, "Die Manometrie der Lungen," *Berliner klin. Wochenschr.*, 1871, No. 45.

[‡] *Deutsches Archiv für klin. Medicin*, vol. XVII; *Berliner klin. Wochenschr.*, 1880.

[§] *Berliner klin. Wochenschr.*, 1879, No. 42.

It was hoped that much information would be obtained from the respiratory tracings, but the sources of error and the difficulties to be overcome in the application of the method are very great, and the data must be accepted with a corresponding degree of reserve. The best exposition of the technic and the possible failures of this method of examination is found in Riegel's work.‡ His stethograph is one of the best of the instruments that have been used for this purpose. The investigations of Vierordt and Ludwig, Ackermann, Gerhardt, and Heimke are equally valuable, but have no bearing on the question of emphysema. In all these methods the movement of a certain point on the wall of the thorax is graphically represented by means of rigid rods, and in Riegel's method the movement of the point is magnified. Other apparatus which record the variations in the volume of the lungs have also been used by numerous investigators. Among these I include especially Marey's pneumograph. If the observations are correctly made, the two methods yield comparable results.

Riegel describes the normal respiratory curve as follows. Inspiration begins relatively slowly, increases quite rapidly, and attains its maximum of velocity approximately at the end of the first third of the entire inspiratory excursion. This velocity is maintained during nearly the whole of the second third, when it begins to diminish and finally falls to the minimum. The transition from the inspiratory to the expiratory limb of the curve is extremely gradual. The expiratory velocity then increases until it attains its maximum, which likewise lasts about one-third of the way, the velocity beginning to decrease during the last third. The transition from expiration to in-

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* "Transactions of the Seventeenth Congress for Clinical Medicine" (Innere Medicin), p. 602.

† *Pflüger's Archiv*, vol. LXXVI, p. 356.

‡ "Die Athembewegungen," Würzburg, 1873.

spiration is very gradual; a respiratory pause at this point may or may not be present. In emphysematous subjects the inspiratory portion of the curve rises more rapidly, the transition to the expiratory limb is also abrupt; the expiratory portion of the curve at first declines rapidly, then the velocity rather suddenly diminishes, and the rest of the expiratory path is covered at a considerably diminished rate of speed. The disproportion between the duration of inspiration and expiration is comparatively great, the latter being considerably prolonged. The time gained by the increased inspiratory velocity is lost by the prolongation of expiration. The number of respiratory movements in the dyspnea of emphysema is not necessarily increased, although an increase takes place in severe grades of the disease.

Other features of the emphysematous curve, such as the occasional presence of an inspiratory pause (pause between inspiration and expiration) and the differences presented by curves taken at the same time from two different points, are less important. The fact that, as the mobility of the thorax diminishes, the differences at certain points become very slight or even reduced to zero, is not particularly characteristic of emphysema. Further details on this point will be found in Riegel's book.

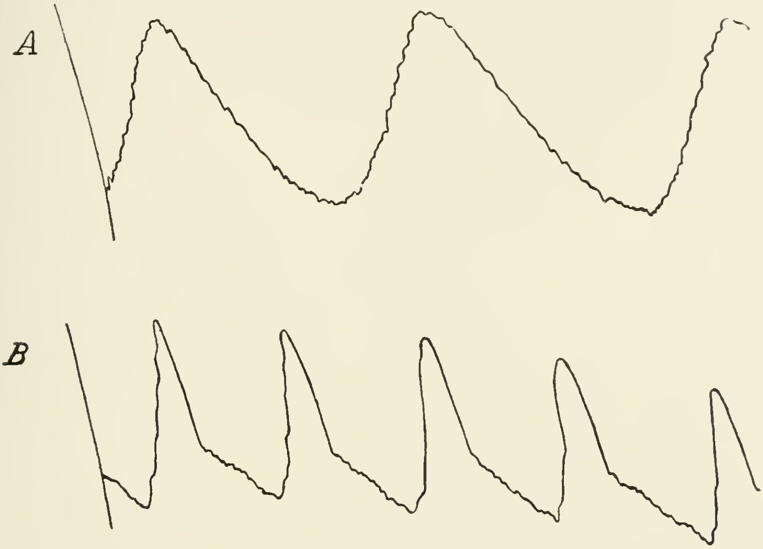

FIG. 12.—Respiratory Tracings.

The two curves in the accompanying diagram from Riegel (Fig. 12) illustrate the description that has just been given. Figure *A* is the normal respiratory curve obtained by placing the stethograph over the sternal insertion of the diaphragm. Figure *B* is the respiratory curve of an emphysematous subject taken at the same point on the thorax.

How easily an entirely different result may be obtained by another method is shown by the recent work of Aron.* He took a Marey's capsule and fastened it about the thorax with a belt. This he connected with a mercury manometer; the capsule and the rubber tube which connects it with the manometer, and the limb of the manometer as far as the column of mercury, were then filled with water. From his experiments, which possess the special advantage of having been performed on sleeping human subjects, he deduces the following description of the normal respiratory curve:

Inspiration proceeds quite uniformly and gradually. The ascending limb is moderately oblique; the apex of the curve is, as a rule, quite acute; that is to say, the transition from the inspiratory to the descending expiratory limb is quite abrupt. The inspiration (distention) consists of two phases that cannot very well be separated one from the other. The first portion of expiration proceeds nearly as rapidly as inspiration, while the latter portion takes place more slowly, a condition that is graphically expressed in the more horizontal portion of the expiratory curve. The transition from expiration to the succeeding inspiration is gradual, a change that is indicated by the obtuse angle in the curve. Expiration occupies a longer period than inspiration.

The same author describes the emphysematous curve as follows: While the inspiratory limb of the curve does not present any marked deviation from the normal, the expiratory curve, on the other hand, is materially modified. This modification shows itself in the shape of the apex, which, as a rule, is broader and more rounded than is observed under any other conditions so far subjected to examination. The entire subsequent expiration is delayed in the same way as the transition from inspiration to expiration. The expiratory curve therefore presents a more oblique continuous line, extending down to the beginning of the succeeding respiration. It is quite impossible to distinguish two separate divisions in the expiratory curve.

It is evident that the difference in the results obtained by these two investigators depends on the difference in the methods employed. The fact that Aron examined sleeping individuals, while Riegel's subjects were awake, appears to me an advantage in favor of the former's accuracy. There is no doubt that respiration may be greatly modified if the individual's attention is directed to his respiratory movements. On the other hand, Riegel examined such a large quantity of material that he must have been fully aware of the importance of this subjective influence. Again, the apparatus of Aron, owing to the column of mercury which it contains, must be subject to considerable intrinsic vibration. The fact that Aron distinguished two separate divisions in normal expiration, which were not described by any author before him, cannot help arousing some misgivings. These two divisions in the respiratory curve Riegel finds only in the emphysematous patient. In order to obtain a better opportunity for judging of the respective accuracy of these results I have examined

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* *Virchow's Archiv*, vol. CXXXVII, p. 179.

other respiratory curves which I found in Gerhardt's "Lehrbuch der Auscultation und Percussion," and in Heimke's Dissertation, Jena, 1865, but unfortunately these curves are insufficiently described and discussed, so that they are not clearly intelligible, and cannot therefore be utilized. Marey's respiratory curves, on the other hand, closely approximate the description given by Aron: Two segments are to be distinguished in the respiratory curve, a more vertical and a more horizontal segment. Marey, however, interprets the horizontal portion not as the product of expiration, but as a respiratory pause, while Aron refuses to admit the existence of a respiratory pause.

The conditions attending the use of these instruments present many sources of error which are usually overlooked, but become prominent on closer examination, as Pick has shown in a paper on the subject.* He observed that the retarded passive expiration or respiratory pause is not immediately followed by inspiration, but that between the two phases there is a short and feeble, so to speak, pre-inspiratory active expiration, effected by contraction of the lateral or median upper portions of the abdomen. This phenomenon is expresesd in the respiratory curves by a more or less abrupt bend at the end of the expiratory limb. He suggests as an explanation that during expiration a certain degree of dyspnea is developed which stimulates the respiratory center at a time when it is still "set" or "tuned" for the emission of an expiratory impulse.

Pick's paper is altogether very instructive, and throws much light on the study and significance of the respiratory curves. He used a Roth polygraph. The elastic sac which serves for the reception of the respiratory movements was placed on the middle of the epigastrium, but when the movements were taken from other points of the body, entirely different tracings were of course obtained. When one sac was placed on the manubrium and the other on the epigastrium, the epigastric curve was found to present a depression at the instant when the beginning of inspiration recorded itself in the upper thoracic tracing by a rise in the curve. The apex of the epigastric curve appeared somewhat delayed in comparison with that of the thoracic curve. This is because marked retraction of the lower portion of the thorax sometimes takes place during inspiration in emphysema and, unless inspection has been very careful, the tracing becomes quite unintelligible.

At all events Riegel has not exhausted the subject of the respiration in emphysema.

In all these observations only certain points on the thoracic wall, especially the anterior wall, were studied. The movement of the entire thorax is such a complicated matter that it has so far been impossible to grasp it, and commit it to paper. Many details still remain to be studied. Whether such a study would be productive of any very remarkable results remains to be decided. It seems at least probable that the various forms of emphysema and the differences in the clinical course have an important bearing and deserve more extensive observation.

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* *Prager med. Wochenschr.*, 1883, No. 17.

In its present state stethography is of no importance in practice, in spite of the interest which this method of examination has aroused.

In the study of the individual inspiratory and expiratory muscles the attention was naturally directed chiefly to the **diaphragm.** The importance of this muscle is not rated so high now as it was formerly, as the external intercostals and the intercartilaginei are winning more and more recognition as important inspiratory agents.* Nevertheless clinical observation of the diaphragm will always maintain its prominence, especially as the strange theory of a special feminine type of respiration is being more and more generally discarded. The relation between the two great inspiratory muscles is in need of further elucidation.

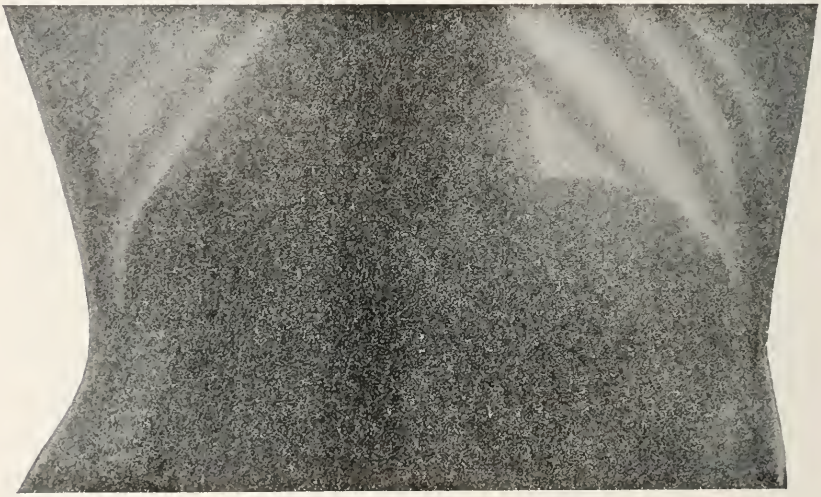

Fig. 13.

The decrease in the elasticity of the lungs and the increase in the quantity of residual air are accompanied by an augmentation in the volume of the organ which has at its disposal the comparatively small complementary spaces in the anterior margin, and the larger complementary spaces at the diaphragm; these are logically utilized in partial emphysema. In diffuse emphysema, however, the case is somewhat different. The characteristic feature of this variety is the great extent of the emphysema. Although its distribution may not be quite uniform, it nevertheless extends diffusely over the entire area of both lungs. The complementary spaces are of little importance, for the lung becomes distended as a whole and the diaphragm is forced downward. The distention can easily be demonstrated by percussion, and is found, as a rule, to amount to two intercostal spaces, and in some regions to still more. In the text-books it is found stated that the diaphragm can be depressed to such a point that the convexity is directed downward and the diaphragm during contraction acts as an expiratory muscle. I believe this is a purely theoretic conclusion, and I do not believe that life can continue if the diaphragm loses its power of acting as an inspiratory muscle.

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* Compare R. Fick, "Ueber die Athemmuskeln," *Archiv für Anatomie und Physiologie*, 1897. Anatomische Abtheilung, Supplement.

Until recently percussion was the only method available for the determination of the position of the diaphragm in the living subject, and that method of examination is clearly inadequate to act as a basis for such a daring assertion. Since I have begun to observe the diaphragms of numerous emphysematous subjects with the Röntgen rays, I have always found the dome of the diaphragm more or less distinctly flattened, but have never seen a perfectly flat diaphragm, far less one with its convexity directed downward.

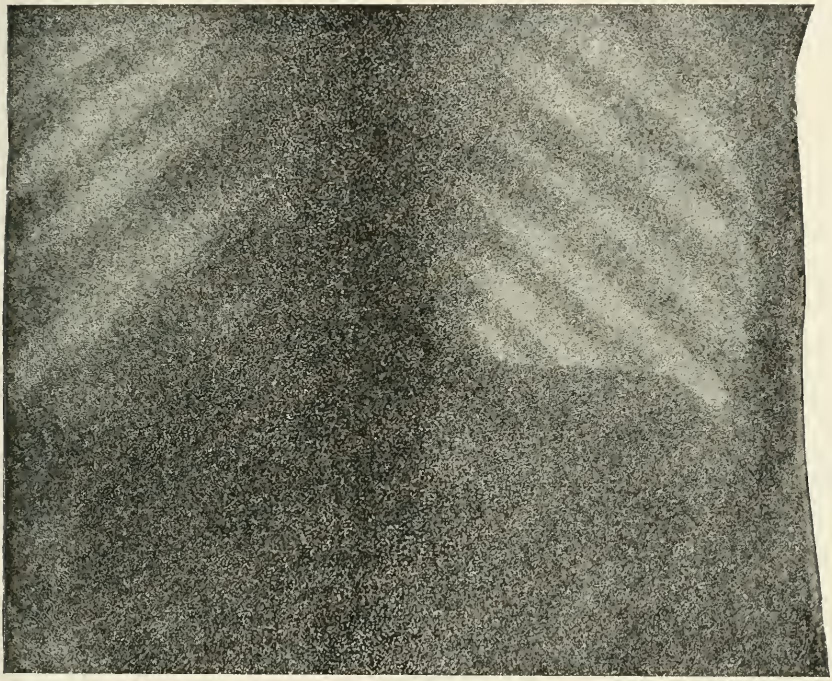


Fig. 14.

The two figures in the text (Figs. 13 and 14) give an excellent illustration, the one of the normal arch of the diaphragm in an elderly man, the other of the flattening of the diaphragm in a case of severe emphysema. I have never seen more flattening than is represented in figure 14. On the other hand, the arch in figure 13 does not by any means represent the highest degree possible under normal conditions, as the heart appears embedded in the diaphragm; in younger individuals the arch is absolutely like a cupola on the top of which the heart is supported.

The diaphragm appears very distinctly in the Röntgen ray image and is no doubt destined to afford the clinician much valuable information for diagnosis. Nevertheless the value of the photograph has been greatly overestimated. In clinical practice observation on the living subject is infinitely more important; the movements of the diaphragm under various influences appear either quiet or excited, the excursions may be great or small.

[The pulmonary area is more extensive and brighter than in health, and reaches not only lower down, but higher up in the chest. The diaphragm is lower down in the thorax, and its excursion is restricted, and is restricted in the upper part of its usual movement. It sometimes happens that the diaphragm is so low down during full inspiration that it has a peculiar outline, this outline being made up of two curves on each side, instead of one, and following the outline of some of the organs directly under it. But though in quiet breathing the diaphragm may be low down in the thorax, it may be brought much higher up in the chest during a forced expiration. This increased excursion may be caused by the upward pressure of the contents of the abdominal cavity, a pressure arising from the contraction of the abdominal wall. The cardiac outline stands out with unusual clearness on the fluorescent screen, as do the other outlines, such as the ribs and clavicles. The heart changes its position far less than usual during deep inspiration; it is lower down in the thorax than in health. Frequently the right ventricle and the right auricle are seen to be enlarged (Williams).—Ed.]

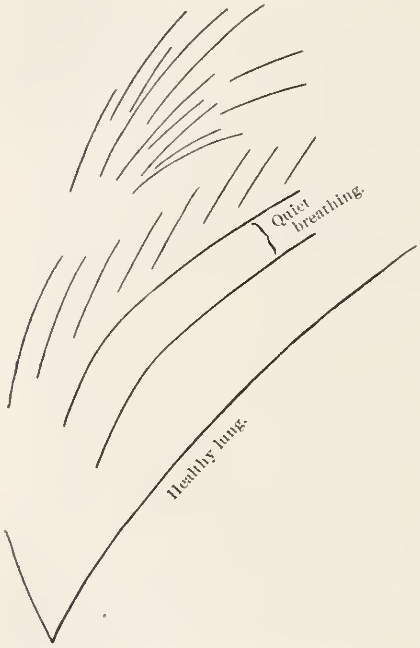
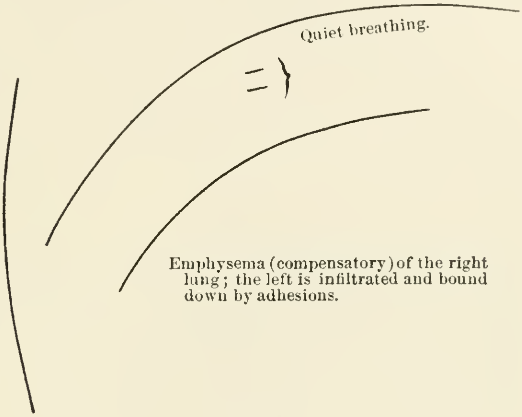


Fig. 15.

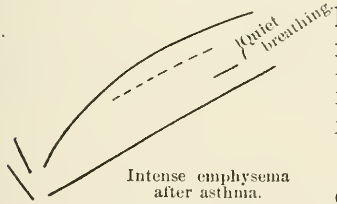
In observing a normal diaphragm with the fluoroscope, especially the right half, which appears more distinctly and in greater extent than the left, the characteristic arch and the rhythmic flattening that accompany the respiratory movements can be readily seen. Respiration is so slow that I have often been able to record the flattening on the instrument which is covered with a glass plate. The inspiratory and expiratory positions of the diaphragm in a normal individual during quiet breathing are shown in the accompanying illustration (Fig. 15).

When the subject is asked to draw a deep breath, the diaphragm becomes sharply outlined during inspiration and appears almost as a straight line; during forced expiration it makes a sudden rise and the entire picture at the same time becomes more and more blurred, so that it is impossible to determine positively at what point the diaphragm became stationary. It could, however, be determined with certainty that with the Röntgen-ray tube at a distance of 60 cm. the excursion of the diaphragm on the screen was about 9 cm., rather more than less. It is really astonishing to witness this enormous excursion. The indistinctness of the image during forced expiration is partly explained by the greater approximation of the ribs, and partly by the fact that the pulmonary tissue becomes greatly consolidated, most of the air being forced out. All the tissues are crowded together, and although, according to Traube's view, a small quantity of air remains in the alveoli, these structures also are greatly contracted, and it is quite comprehensible that the lung ceases to be transparent during a forced expiration. In figure 15 the lowest line indicates the flattening of the diaphragm during very deep inspiration, the two upper lines indicate the excursion during quiet respiration, while the shading in the upper part of the picture is intended to represent the obscuration and blurring of the diaphragm during forced expiration.



Emphysema (compensatory) of the right lung; the left is infiltrated and bound down by adhesions.

Fig. 16.



Intense emphysema after asthma.

Fig. 17.

This loss of transparency on the part of the lung and the blurring of the line of the diaphragm appear to me characteristic of a perfectly normal, vigorous, and youthful condition of the parts. In older individuals, even though they have not a trace of emphysema, and in younger individuals suffering from any alteration in the lung, the phenomenon ceases to be so well marked. The upper limits of the diaphragm in such cases are quite distinctly seen. A difference of 7 cm. between the upper and lower lines, that corresponding to the deepest inspiration and that corresponding to forced expiration, with the tube at a distance of 60 cm., I regard as within the limits of the normal in adults.

If we now turn to the diaphragm in an emphysematous subject (see Figs. 16 and 17), we find an altogether different condition. The difference is not such as has been described on theoretic grounds, however, for the diaphragm is flat and does not arch downward; on the contrary, it presents, as in the healthy subject, a distinct downward concavity which diminishes during inspiration. During quiet breathing there is an astonishingly small difference between inspiration and expiration. The curve is no doubt somewhat flatter than in health, but the difference is very variable; it would be a daring and presumptuous undertaking to attempt to determine from the degree of convexity whether the diaphragm in question was that of an emphysematous, or of a healthy subject. When, however, the patient is asked to breathe deeply, it is astonishing to see how much less such a patient is capable of than a healthy individual. During deep inspiration the diaphragm, it is true, also attains the horizontal line, and during forced expiration it rises somewhat. But the lung does not become obscured, the shadow of the diaphragm remains perfectly clear and distinct, and the excursion on the screen, with the tube at a distance of 60 cm., is always less than 7 cm.; often it is not more than 2 or 3 cm.

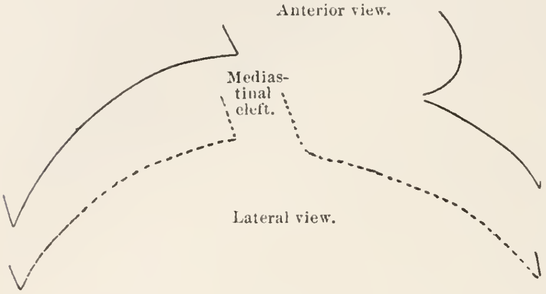


Fig. 18.

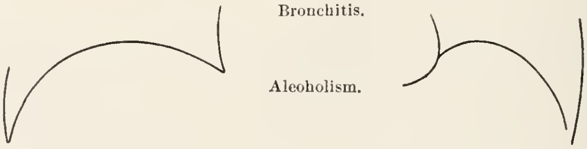


Fig. 19.

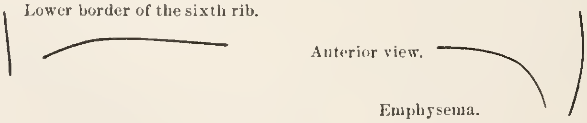
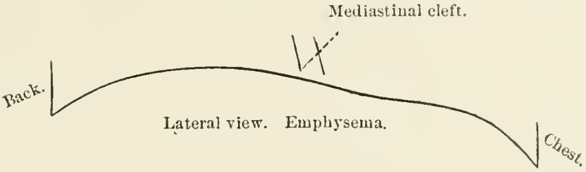


Fig. 20.

In the accompanying figures the beginning and end of the diaphragmatic line are not shown, as the drawing presents only what can be recognized with absolute certainty. The modification in the concavity of the arch, and the excursion, are the points that are most conspicuous and that can be determined with the greatest degree of certainty. The depression of the centrum tendineum is probably not great. At the apex of the heart it was impossible to demonstrate any inspiratory depression.

Figure 20 represents the position of the diaphragm in a case of very severe emphysema in which no movement at all could be seen during quiet respiration; it is evident that the right half of the arch is much less curved than the left. This is a common phenomenon, and calls for further observation.

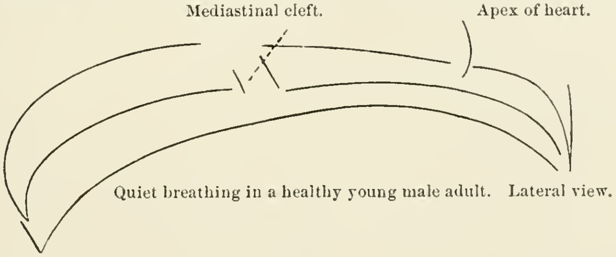


Lateral view. Emphysema.

FIG. 21.

The behavior of the diaphragm with respect to the heart is shown in the outlines of figure 18. This figure represents the normal behavior in a youthful, healthy, and vigorous individual. The dome-like arch, above which the heart hangs unsupported, is very well shown. An interesting contrast is represented in figure 19, which is taken from an elderly drinker who had not as yet developed a severe degree of emphysema. In this figure the heart appears to lie within an excavation in the diaphragm; but an excavation of this kind is so frequently seen that it undoubtedly belongs to the realm of the physiologic.

In the lateral view, which shows the Criegern mediastinal cleft, the diaphragm and the flattening of its dome can also be very satisfactorily studied. In fact, the lateral view presents many advantages. Figure 21 represents the diaphragmatic line of the same emphysematous subject whose thorax is shown in anterior view in figure 20. Figure 22 shows the relations in the healthy subject; the intermediate line represents the median position from which the usual oscillations take place to the upper and lower lines. The lower line corresponds to a forcible expiration, the upper to a moderately forcible expiration, not great enough to bring about blurring of the diaphragmatic line.



Quiet breathing in a healthy young male adult. Lateral view.

FIG. 22.

The largest excursions as measured on the screen, with the tube at a distance of 60 cm., and with the chest of the subject in contact with the screen, are as follows:

Fig. 15. Quiet breathing 1.7 cm.
" 16. " " 0.4 " Largest excursion 3.2.
" 71. " " 1.1 " Largest excursion 2.9.
" 22. Greatest distance 6.1 cm.: immediately in front of the apex of the heart, 3; behind the mediastinal cleft, 5.

The exact position of the dome of the diaphragm can also be determined on the body of the living subject with the aid of transillumination. The patient must, of course, be placed as near the screen as possible and the tube must be adjusted at the proper level. If the tube is too high or too low, the image will also be projected too low or too high. The proper position for the tube can be determined without much difficulty. In this way not only can the line of the diaphragm be traced on the glass plate of the screen, but, by introducing the pencil between the patient's body and the screen, the height of the diaphragmatic dome can be traced on the chest-wall. In the healthy subject this line is found in the anterior right mammillary line at the lower edge of the fifth rib or in the fifth intercostal space. On the left side the line is a shade lower. The usual statement that the diaphragm only extends as far as the fifth rib is based on postmortem findings, and cannot therefore be accepted as correct in the living subject. Frerichs appears to be right, therefore, when he says that "the true upper limit of the liver is in the mammary line, usually in the fifth intercostal space."*

In emphysema the arch of the diaphragm in most cases is somewhat lower—on the sixth rib, or below the sixth rib in the sixth intercostal space, rarely on the seventh rib; I have never seen it lower than this point (in the mammary line).

In ordinary practice the determination of the position of the diaphragmatic dome is not practicable, nor would it be of any special utility. Percussion is too uncertain a method of determining the so-called relative upper boundary of the liver with accuracy, and in emphysema the difficulties are increased. For practical purposes it is quite enough to determine on the right side the line where absolute liver dulness begins, and on the left side, by means of deep percussion, the boundary between pulmonary resonance and gastric tympany. This boundary is, as a rule, very distinct. The position of these two lines both on the right and on the left side is always lower (in emphysema) than in health. The boundary of the liver is generally given as the sixth rib in the mammary line, the eighth rib in the axillary line, and the eleventh rib at the vertebral column. In emphysema the boundary in front is from one to two ribs lower in the axillary line. The difference rarely amounts to two ribs, and at the vertebral column it is at most one rib. This is because the complementary space is lowest in front and gradually diminishes in depth toward the vertebral column. The anatomists tell us that the insertion of the diaphragm is found in the line which passes from the seventh costal cartilage to the tip of the twelfth rib. In the mammary line the insertion is at the eighth rib, in the axillary line at the tenth, and at the vertebral column at the twelfth. These points of insertion evidently determine the line that we are able to define by percussion in the highest grades of emphysema (see Percussion).

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* "Klinik der Leberkrankheiten," I, p. 33.

In conditions of marked dyspnea the lower portion of the thoracic wall, and with it the insertion of the diaphragm, as is well known, is greatly retracted during inspiration; this retraction is most distinctly visible on the right and on the left side in the mammary line, but the lower portions of the sternum and the lateral portions of the thorax may also be involved. The usual explanation—that the external air-pressure, owing to the slower entrance of the air into the lung during inspiration, gains the upper hand and exerts its influence especially on the points referred to, where the contracting diaphragm at the same time exerts countertraction, while it is prevented by the rarefaction of the air in the thoracic space from descending—is generally recognized and is fully discussed by Gerhardt in his "Stand des Diaphragmas." He also established the fact that an inspiratory rise of the diaphragm can be determined under such circumstances by observing the lower border of the liver, but for my part I have never been able to demonstrate this phenomenon incontrovertibly in emphysema.

Litten's Sign.—A very valuable and beautiful symptom in the determination of the movements of the diaphragm is due to the discovery of Litten. In any healthy individual a furrow can be seen on both sides of the body, chiefly in the axillary line, which becomes distinct a little after the beginning of a forced inspiration between the fifth to the eighth intercostal spaces and moves upward a distance of from 5 to 6 cm. during inspiration. In well-developed cases of emphysema this phenomenon is regularly absent; in mild grades of the disease faint traces of the furrow with small excursions can sometimes be recognized.

Nothing is known of the action and behavior of the *interossei* in emphysema. Since the action of the diaphragm is manifestly and undoubtedly impaired, it might be expected, according to the view that the external interossei and intercartilaginei represent powerful inspiratory muscles, that the activity of these muscles should be correspondingly accentuated. It is certain at least that the intercostal spaces do not bulge in emphysematous subjects, as might be expected from the increase in the volume of the lungs. During the act of coughing, it is true, they are driven outward, especially along the sternum in the upper intercostal spaces. Beyond the domain of the external *intercostals*, slight depressions are seen even during deep inspiration, and if the finger is applied to one of these depressions, the hardening of the outer margin of the depression which is formed by the external intercostal can be distinctly felt (Schmidtlein). The *scaleni* and *sternocleidomastoid* muscles in such patients usually become visible as hypertrophied cords, and contribute materially to the characteristic appearance of emphysema. In some patients the sternocleidomastoid muscles, in others the scaleni, appear more prominent. The mere prominence of the muscle alone is not a reliable sign unless the hardening can also be distinctly felt. Schmidtlein, who made a special study of these matters, says that the difference depends on whether the first rib is ossified and the sternocostal articulation obliterated. When that is the case, the sternocleidomastoids become more active, while in the opposite condition the scaleni contract more vigorously. In the worst cases the action of both muscles no doubt becomes accentuated. The *levatores costarum* muscles cannot be observed in the living subject. They assist in bringing about extension of the vertebral column which accompanies every complete expiratory movement. The notion that they elevate the ribs is now practically abandoned.

The *pectoralis minor* also represents an inspiratory muscle when the scapula is fixed, and this is true in an even more marked degree of the more powerful serratus anticus major. This is why emphysematous patients are so fond of fixing the shoulder girdle by leaning on their elbows when they are sitting down, as by this means the costal insertion of the latissimus dorsi is made available during inspiration. Another important advantage gained by leaning on the elbows is that suggested by R. Fick. The movements of the ribs are impeded by the pressure of the shoulder girdle which is suspended to the vertebral column by the levator scapuli, the trapezius, and the rhomboid muscles; and the movement of the ribs is facilitated when the scapula and the clavicle are pushed upward from the ribs through the agency of some external support.

The development of the *pectoralis major* is not particularly favored in emphysema, because it becomes an effective respiratory muscle only when the upper arm is fixed in extension. Hence the arms are raised above the head in artificial respiration. The muscle, however, is probably of some assistance even when the arm is supported on the elbow.

The *serratus posticus superior* and the *ileocostalis cervicis* cannot be seen. The *serratus posticus inferior* is visible, but is of so little importance that it is not taken into consideration. Many physiologists regard it as an expiratory muscle.*

Among the **expiratory muscles** the *rectus abdominis* shows the greatest degree of development, although all the other abdominal muscles are well marked and distinctly hard on palpation. The *external oblique* draws the seven lower ribs directly downward; and the remaining ribs, by compressing the contents of the abdomen, force the latter to rise and push the dome of the diaphragm before them. The expiratory muscles in the back are for the most part invisible, and inaccessible even to palpation; they are the *ileocostalis lumborum*, the *quadratus lumborum*, and part of the *longissimus dorsi.*

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* See Landerer, "Archiv für Anatomie und Physiologie," 1880.

In severe degrees of emphysema it is often observed that the rectus abdominis, and sometimes even the external oblique and transversalis muscles, become converted from expiratory to inspiratory muscles; *i. e.*, they undergo a distinct contraction at each inspiration. It is probable that in this way they assist the action of the diaphragm, for whenever the abdominal viscera are forcibly compressed they offer so great a resistance to the descent of the diaphragm as to enable that muscle to enlarge the lower aperture of the thorax. To do this, the diaphragm must be able to participate in the elevation of the six lower ribs, for the ribs in moving upward must, owing to the peculiar shape of their articulations, at the same time move outward. Elevation of the ribs by the diaphragm is possible only when its fibers are so placed that in contracting they elevate the ribs. If, therefore, the diaphragm is forced upward by the pressure of the abdominal viscera, the direction of these fibers becomes more and more favorable (vertical); in fact, if the diaphragm is normally placed low, it will be unable to elevate the ribs except under these favorable conditions. Sappey believes that such an action of the diaphragm takes place even in normal individuals. There is no doubt that any individual has it in his power by exerting his will: (1) To breathe in the ordinary manner—*i. e.*, arching the epigastrium, raising and arching the ribs somewhat laterally so that the upper thoracic space is little enlarged, but becomes elevated as a whole; (2) to breathe in such a manner that the epigastrium becomes depressed and the upper portion of the thoracic space undergoes greater dilatation and elevation, while the lower ribs also are elevated, but are chiefly forced outward.* In the second type of breathing the diaphragm acts in the manner above described. This type has been especially, but incorrectly, described as the feminine type of breathing. Any garment that constricts the lower opening of the thorax must, of course, favor its production, and a tightly laced corset undoubtedly has this effect, but so has any coat that is well "cut in at the waist," as, for example, a military coat. But the skirt band which rests on the hips evidently does not produce such an effect. The movement of the sternum must also be taken into consideration. At each inspiration the first rib and the sternum are raised, and as the ribs increase in length from above downward and the arch becomes greater, the sternum must move forward, rotating about an axis which passes in the frontal plane through the two insertions of the first rib. The lower portion of the sternum therefore moves further away from the vertebral column than the upper portion, while the upper extremity of the bone practically remains immovable. The more vigorous the action of the auxiliary muscles of respiration, the greater the elevation of the thoracic cage as a whole. Ordinary vigorous inspiration is accompanied only by moderate elevation of the first rib and of the clavicle, and while the elevation of the lower ribs is greater, it is accompanied by a forward movement, so that the width of the intercostal spaces remains approximately the same. Landerer found in the cadaver that the three first intercostal spaces contract during inspiration while the lower spaces become somewhat wider. The thorax as a whole was about 2 cm. longer in the inspiratory, than in the expiratory position, but this does not necessarily apply to the living subject.

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*Landerer has attempted to explain why the upper ribs show a greater aptness and tendency to become elevated, while the lower are more apt to move outward.

The normal thorax possesses a certain *resiliency*, by virtue of which it constantly tends to regain its position of rest. But what is this position of rest? It was formerly thought that the position of rest corresponded to the end of inspiration. Henke then taught that the equilibrium of the ligamentary apparatus of the thorax was represented by the inspiratory position and that the resiliency of the thorax could act for a short space of time as an expiratory force only during very forcible inspiration.* Under ordinary conditions (according to Henke) the thorax, by virtue of the elasticity of the costal cartilages, returns to a position of elevation as its normal position of rest. It appears to me rather suspicious, however, that he fails to give any good reasons for this statement. He says: "This position of rest is indicated by the anterior extremity of the rib, its junction with the sternum being in the plane of its axis of rotation." He reached this conclusion through observations made on moist preparations. He acknowledges, however, that torsion of the costal cartilages, the effects of which are not quite so easy to interpret, also enters into the problem. Henke's view finds support in an investigation made by Landerer.† He set a cadaver on its feet, punctured the pleural sacs, cleared out the intercostal spaces, separated the anterior mediastinum from the sternum, and sawed through the manubrium and sternum crosswise between costal insertions, beginning above. After the manubrium had been divided with a saw between the first and second ribs, the upper portion with the first costal insertion moved upward 10 mm. and outward 4 mm. The lower portion descended 4 mm. As he continued to saw through the sternum downward between two ribs, the portion attached to the abdomen descended more and more. The upward and outward movement was most marked at the first and second costal insertions and disappeared altogether at the sixth. From this and other investigations, which space forbids describing here, he arrived at the following result: The position of equilibrium of the thorax is the product of the elastic tendency of the six upper pairs of ribs to move upward, and the weight of the thorax and abdominal viscera which counteracts that tendency. The elasticity of the upper portion of the thoracic wall therefore assists in inspiration in every position of the body, but most in the horizontal position, in which it is least antagonized by the weight of the abdominal viscera. Inspiration, therefore, is first affected by loss of elasticity in the thorax. The elasticity of the lungs, on the other hand, evidently favors expiration, hence expiration is first effected by a loss of pulmonary elasticity. When the two factors coexist in varying degrees, and react one on the other, it is at once evident that a great variety of results are possible.

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* "Handbuch der Anatomie und Mechanik der Gelenke," p. 86.

† "Archiv für Anatomie und Physiologie," Physiologische Abtheilung, 1881, p. 278.

The position of the body is not unimportant, for in the erect position the weight of the abdominal wall and of the thorax tends to assist expiration, while in the horizontal position this expiratory aid is altogether wanting. Hence the resultant of all the forces that act on the thorax will vary in the different positions of the body; the horizontal and sitting position tend to favor inspiration, while the erect position favors expiration.

It follows that the expiratory muscles must overcome a certain resiliency on the part of the thorax, and force it out of its normal position so as to diminish its volume. The younger the individual, the more effective will be the respiratory musculature in this respect. In old age this action on the part of the muscles is scarcely possible. As the costal cartilages become calcified, the forward movement of the sternum is rapidly impaired, while the upward movement remains and becomes more prominent than ever. The upward movement is not, however, accomplished by the resiliency of the thorax, and an extra amount of work is therefore thrown on the inspiratory muscles. The thorax is laboriously elevated as a whole and then falls again like lead. If the lung is atrophic, the thorax often does not appear dilated and the intercostal spaces may even be depressed. Such elevation without corresponding distention is a sign of calcification of the cartilages and obliteration of the sternocostal articulations, and these conditions are so common in emphysema that they may be regarded as signs of that disease. But the explanation cannot be applied without qualification to young emphysematous subjects.

Youthful individuals have a beautifully arched thorax corresponding to its inspiratory position; the thorax, it is true, neither expands nor collapses sufficiently, but it does not present the rigid appearance observed in old persons, in whom the thorax moves as a whole, or like a solid mass, as it were. The resiliency of the upper ribs is preserved, the inspiratory muscles are not forced into excessive exertion, and their development as a matter of observation is not nearly so marked as would be expected from the high degree of cyanosis. Accordingly, Traube observed in youthful individuals suffering from heart disease an abnormally arched thorax, corresponding in its chief characteristics to the emphysematous thorax. He attributes the shape of the thorax exclusively to the inspiratory efforts, which are quite different in such individuals after even moderate exercise.

The study of the *absorption of oxygen* and the *elimination of carbon dioxid* has not been neglected. *A priori* it was to be presumed that both would be increased. Geppert* studied the question on three emphysematous subjects. He discovered the remarkable fact that the degree of ventilation was not materially different from that which takes place in the healthy subject. The latter he estimates at 8.3 to 10 liters per minute, while in emphysematous subjects the corresponding figures are 8.33 to 11.76 liters; if there is any increase, therefore, it is very slight. As regards the composition of the expired air, he calculates for the healthy subject, from the analyses of earlier investigators, 4.1 to 4.38 of carbon dioxid, and 16.033 to 16.28 of oxygen, making a deficit of 4.8 to 5.1 of oxygen. In his emphysematous subjects he obtained 2.32 to 2.6 carbon dioxid and 17.48 to 18.44 oxygen, making a deficit of 2.6 to 4.2 oxygen. The low figures he obtained for the absolute elimination of carbon dioxid are in themselves sufficiently remarkable.† The behavior of the expired air is the same as in excessive ventilation; *i. e.*, parts of the circulatory system in the lung which are accessible to the air are excessively ventilated, while the pressure of carbon dioxid is undoubtedly reduced. The interpretation of this observation is not altogether clear, and suggests the necessity of further investigation.

In conclusion, I must not omit to mention the interesting fact that Traube was able to confirm the statement‡ that pulmonary emphysema in itself is quite incapable of producing dyspnea. He concludes that the slow destructive process in the lung can produce only a gradual decrease in the supply of oxygen and formation of carbon dioxid; but when so little carbon dioxid is produced, the amount necessary for the production of severe dyspnea cannot easily accumulate in the blood of an emphysematous subject. Since, therefore, the small amount of carbon dioxid in the inspired air has been quantitatively determined by Geppert, it follows, does it not, that the carbon dioxid tension in the blood is also low? But this is a rash method of reasoning. The tension may even be increased if, for example, some other obstacles to the interchange of gases are present; and that they are present can hardly be doubted. It is a bold assertion on Traube's part that the production of carbon dioxid must diminish on account of the diminished supply of oxygen (evidently through the lung), since the two values are, to a great extent at least, independent of one another.

2. In addition to simple dyspnea, distinct **dyspneic attacks** also occur in emphysema. Some of these are undoubtedly attacks of true bronchial asthma, at least in cases of the asthmatic form of emphysema. Another group of cases belonging to cardiac asthma may be seen in their most pronounced form in patients suffering from cardiac emphysema. As, however, the heart sooner or later becomes involved

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* *Charité-Annalen*, IX, 1884, p. 282. Compare also Möller, *Zeitschr. für Biologie*, vol. XIV.

† He agrees with Gréhant, *Gazette des hôpitaux*, 1880.

‡ "Gesammelte Abhandlungen," III, p. 413.

in every emphysematous individual, cardiac asthma must be regarded as a true symptom of emphysema, just as, conversely, emphysema may be superadded to cardiac asthma developing from other causes. I do not believe there is any justification for distinguishing any special dyspneic attacks independent of asthma or cardiac weakness in addition to the two varieties here described.

3. After dyspnea, **enlargement of the lung** is the most important symptom of emphysema. It is demonstrated by the position of the diaphragm, the distention of the thorax, the displacement of the heart,—signs that have been discussed elsewhere,—and by the protrusion of the lung above the clavicles. The latter sign cannot be produced, however, unless the muscles and fascia that cover in the superior aperture of the thorax have become atrophied from want of nutrition. The sign is therefore not observed in youthful and vigorous subjects, while it is occasionally very pronounced in old and cachectic patients.

In the normal state the arches formed by the lungs and pleuræ project above the oblique plane of the first rib*; the arch formed by the pleura is separated from that corresponding to the lung by a transverse depression due to the subclavian artery: behind is a small but pronounced bulging, and in front the extent of the arch is greater, but the bulging is less prominent. These arches project above the anterior extremity of the first rib, where the upper margin of the sternum extends to the boundary between the second and third thoracic vertebræ—on the average, about 3.5 cm., the figures ranging between 2.5 and 5.5. According to Pansch, no constant difference exists between the right and left sides. The anterior border of the neck of the first rib, which marks the upper limit of the excursion of the pleura in health, is readily determined.

The upper pole of the pleural sac, situated behind the subclavian artery, is almost completely covered by denser tissues, the scaleni muscles, and the brachial plexus; while the anterior bulging is in part covered with loose tissues except the sternocleidomastoid muscle and the clavicle. During deep inspiration the vertical elevation of the apex of the lung above the sternal portion of the clavicle is diminished; although the apex does not alter its position, the clavicle rises. This change is well seen in the Röntgen-ray picture. Pansch is therefore right in refuting the statements of Rüdiger, Heytl, and Guttmann.

In emphysema the portion lying in front of the subclavian artery, which has no immediate covering of solid tissue, is forced forward toward the supraclavicular fossa, between the scalenus and the sternocleidomastoid muscles. In this way the evanescent tumors above the clavicles observed during violent coughing efforts are produced. A simultaneous swelling of the jugular vein sometimes tends still further to increase the size of the tumor. The lower border of the lungs will be discussed under the head of Percussion.

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* Pansch, "Archiv für Anatomie und Physiologie," Anatomische Theil, 1881.

In rare cases the lung is protruded in other regions. The literature of the so-called *pulmonary hernias* has been discussed by Strübing.* The case reported by Friedreich† has now become classic. During a violent attack of cough in an emphysematous subject a tumor the size of an egg suddenly appeared in the fifth intercostal space in the mammary line. The tumor was elastic to the touch and gave out a distinctly resonant sound on percussion. Schmidtlein‡ performed the autopsy on a subject in whom the upper pole of the pleural sac protruded like a hernia half-way up the neck; the hernial sac contained an extremely emphysematous portion of lung. Knox§ reports another extreme case that came to autopsy, as follows: The left lung and the anterior border of the right lung were emphysematous. The apex of the right lung was forced into the neck behind the clavicle and converted into a transparent tumor the size of a man's fist. All traces of alveolar structure had disappeared. After the lung had been removed, the entire hand could be passed up into the neck behind the clavicle. The heart muscle was pale and flaccid, the left ventricle was hypertrophied, the liver and kidneys were in a state of passive congestion.

4. The **shape of the thorax** presents characteristic alterations. In uncomplicated emphysema the thorax is enlarged, the enlargement appearing most marked in the upper half. This impression is accentuated by the fact that the thorax, as a rule, is higher than in the normal state. The fact that the upper anterior portions of the lungs are especially enlarged has been explained by assuming that the catarrh is usually most severe in the lower portions, and that the air drawn in during inspiration can only enter the large air-passages and upper portions of the thorax, but finds an insuperable obstacle in the small bronchi of the lower portions, hence the upper portions of the thorax become more enlarged than the lower portions. The action of the inspiratory muscles is evidently controlled by the need of the body for oxygen. If the lower portions of the lung are less accessible to the air, air hunger will be increased; the diaphragm and other inspiratory muscles are stimulated to greater activity, but the work performed by 'the diaphragm will be relatively diminished on account of the obstruction, and the upper portion of the thorax, which gives attachment to the remaining inspiratory muscles, is forced to perform a correspondingly greater amount of work. In this form of catarrh Riegel actually found with the stethograph that the excursion of the upper portion as compared with that of the lower portion was considerably increased. Riegel points out that in old men whose thoracic walls have lost some of their elasticity the movements of the ribs are materially diminished and the usual excessive dilatation of

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* *Virchow's Archiv*, vol. CXVI.

† "Verhandlungen der physikalisch-medicinischen Gesellschaft zu Würzburg," vol. x, p. 191.

‡ "Deutsche Klinik," 1864.

§ *Lancet*, 1885.

the upper portion of the thorax does not take place. While, therefore, under such conditions, the increased enlargement of the upper part of the thorax may be explained by the mechanism of inspiration, it is interesting to find that Mendelssohn and others utilized this very phenomenon to support their expiration theory. Mendelssohn explained that during forced expiration the lower portion of the lung suffers the greatest compression, and that thus the upward expiratory stream of air must be produced. This expiratory stream meets with the resistance of the air coming from the upper lobe; the pressure is thus increased and the alveoli of the upper portion of the lung are distended.

This pressure is still more heightened when the escape of air through the narrow glottis is interfered with, as usually happens during cough. The air which is compressed in the lower lobes of the lung is forced upward and thus enters the main bronchus of the upper lobe which is directed obliquely downward. I believe that both inspiration and expiration may under certain conditions develop injurious influences in varying degrees: the inspiratory theory cannot be accepted for the cases in which the lower portions of the lung are quite free and accessible to the air, but as catarrh and emphysema are so closely related, the theory cannot be altogether overlooked. The expiration theory also presupposes an injury calculated to produce an enormous increase in expiration, since normal expiration can do no harm. In other words, there must be some morbific or, more correctly, cough-exciting factor, and this is found most frequently in catarrh, which forces us to include in the explanation of the pathogenesis the injury produced by inspiration.

After this little digression, let us return to the consideration of the thorax. The ribs of an emphysematous subject appear excessively arched because they are always seen in the inspiratory position. The sternal extremities of the clavicles are displaced forward in accordance with the position of the sternum. The latter is not protruded beyond the level of the ribs; it is pushed forward along with the anterior extremities of the ribs to which it is attached. It is sometimes described as projecting in the form of a wedge. This phenomenon does not belong to emphysema, but depends on other conditions. As the cartilages gradually become ossified and quite rigid, an arched dilatation is produced. The sagittal diameter is greatly increased, as are also the transverse diameters. The lower opening of the thorax is enlarged so that the ribs where they leave the sternum do not form a right angle, as in the normal state, but an angle equivalent to about 135 degrees. The upper ribs are more horizontal because they are elevated. The intercostal spaces are wide and hard. The distention and forward bulging is generally greater in the upper than in the lower ribs, although there are numerous transitional cases. In the emphysema of old persons the differences gradually disappear. In those cases in which the distention is fairly uniform in the entire thorax, the expression "barrel-shaped chest" is justifiable.

The term barrel-shaped chest is used as if it were a very simple matter, and it is evident that no clear idea of its meaning is, as a rule, associated with the expression. It is really a poor expression, for the ribs are not uniformly arched; they present conspicuous angulations at various points. The normal curve, the apogee of which is marked by the axillary line, is converted into a plane with somewhat rounded anterior and posterior edges, and the walls of the thorax can be quite distinctly subdivided into an anterior, a posterior, and lateral walls. The thorax, when seen from without, thus presents a square shape, and, as the sagittal diameter constantly tends to approach in length the transverse diameter, the horizontal section of the thorax ultimately becomes practically square. The impression of a barrel-shape is especially obtained from the upper portion of the thorax when the arms hang down by the sides and conceal the lateral surfaces. Personally, I think the comparison is a very unfortunate one. When the thorax is arched from above outward, particularly when there is no well-defined peripneumonic depression (Harrison) and the anteroposterior and transverse diameters measured on the outside of the body in the same plane are approximately equal,—they are never quite equal, the transverse diameter is always a little larger than the anteroposterior,—and, finally, when the arch of the upper portion in front corresponds to an arch behind at the level of the shoulder-blades—when these conditions are in evidence, the term barrel-shaped may be employed. It is seen in its most typical form in individuals between the ages of thirty and forty.

The characteristic alterations of the thorax may be absent if the emphysema develops in old age, when the thorax has already become rigid. In fact, the change in the thorax always depends largely on the time of life when the disease begins. The differences in this respect are discussed at some length by Sibson.* It seems to me that emphysema acquired in youth is particularly characterized by the so-called peripneumonic furrow and by the circumstance that the lower ribs, from the sixth to the tenth, appear depressed as compared with the upper ribs. Senile emphysema, on the other hand, is characterized by marked curvature of the vertebral column, protrusion of the head, and a general rounding of the shoulders. Steffen,† in his minute description of the emphysematous thorax in the child, says that it never acquires the barrel-shape, and in regard to the respiration he lays special stress on the fact that only the upper half of the thorax expands during inspiration, the lower half, on the contrary, being retracted. But this is merely an intensified form of the phenomenon observed in all emphysematous subjects, because the excessive contraction of the inspiratory muscles especially affects the upper ribs.

Fürst ‡ also asserts that the thorax in young children never, and in older children very rarely, presents the barrel-shape, and that it is only when the thorax is deformed as a result of rachitis or abnormally early ossification that the shape somewhat suggests that of a barrel. These statements do not in the least agree with my view; for in these subjects the portions about the nipple are contracted and the lower costal border projects outward in the shape of a shovel, which does not in the least suggest a barrel, although it is true that the arch of the back is well marked. The dilatation in the anteroposterior direction, Fürst says, is fairly constant (personally, I should be inclined to call it quite constant), and affects especially the upper portion of the thorax, where the oblique diameters are also enlarged. Sometimes the supraclavicular region becomes permanently protruding. Below the fourth rib the thoracic space may be found smaller than normal from the presence of the peripneumonic furrow; the thorax is less immovable than in the adult; the breathing in a healthy child is more diaphragmatic, while in an emphysematous child the costal type is more pronounced. Fürst regards expiratory distention, especially of the upper portions of the thorax in the acromial division of the supraclavicular region during violent paroxysms of cough, as particularly characteristic. If the disease is protracted, the neck becomes shortened and the accessory muscles of respiration, the sternocleidomastoid and the scaleni muscles, hypertrophy.

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* *London Med. Gaz.*, 1848, 1849.

† "Klinik der Kinderkrankheiten."

‡ Gerhardt's "Handbuch der Kinderkrankheiten," vol. III.

5. **Curvature of the spine** usually exists, and produces a well-marked arch with its convexity presented outward, the point of greatest curvature corresponding to the lower portion of the thoracic spine. The sternum forms a corresponding but less prominent arch, with its convexity directed forward, the point of greatest curvature corresponding to the insertion of the fourth rib. The angle at the second rib (Ludwig's angle) is very variable; sometimes it is very inconspicuous, and may even be quite obliterated.

The extreme curvature of the vertebral column is accompanied by a bent-over attitude, and the subject of emphysema gradually loses in stature (Sibson). This attitude assists the action of the sternocleidomastoid and scaleni muscles, inasmuch as their insertions on the skull and on the vertebral column occupy a position more directly over their insertions on the clavicle, the sternum, and the ribs, and the muscles are thus enabled to contract in a more effective direction.

6. In regard to the **shape of the sternum,** which is so very conspicuous, it may be said that it appears unusually flat and broad. The so-called sternal angle has long occupied the attention of observers; it appears more or less pronounced in many people, and is caused by the fact that under certain circumstances the plane of the manubrium and that of the gladiolus form an angle with one another. The apex of the angle corresponds to a transverse ridge between the insertions of the second pair of ribs. The mechanism concerned in the production of this angle has recently been investigated by Roths-

child.* The manubrium articulates with the gladiolus by means of an elastic joint, permitting the convexity of the bone to be increased at each inspiration. The manubrium maintains its freedom of movement until advanced age, between sixty and seventy years of age, when its articulation with the gladiolus becomes ossified. The first rib is joined to the manubrium without the formation of a sternocostal joint, the manubrium and the first pair of ribs forming a solid bony ring. Since elevation of the ribs is accompanied by rotation about their long axis, so that the lower border is everted, the movement must be transmitted from the first rib to the manubrium. The movement of the second to the seventh ribs is not communicated to the sternum, being arrested in the articulations that connect the costal cartilage of these ribs with the sternum.† With the aid of an instrument known as the sternogoniometer Rothschild found the size of the normal sternal angle in men to be 15.85 and in women 12.85. During deep inspiration and expiration, an excursion of 14 degrees in men and 12.2 in women was determined. In emphysematous subjects, on the other hand, an abnormally large angle was found between the gladiolus and the manubrium—25 to 33 degrees, as against the normal measurement of 24 degrees in deepest inspiration, although, on the other hand, the excursion was in every instance diminished and in a few cases altogether lost. When phthisis is associated with emphysema of the portions of the lung underlying the sternum, if the articulation between the gladiolus and the manubrium is preserved, the sternal angle may become greatly enlarged, although this is a very exceptional occurrence (this last sentence seems to me obscure).

Rothschild's explanations of the anatomy of the sternum tally with those of the older anatomists, which will be found set forth at length in Freund's work. Rothschild's explanation of the movement of the manubrium, on the other hand, is the exact opposite of that given by Freund,‡ who says: "The first ribs, during elevation, communicate to the manubrium a movement which tends to increase its inclination, so that the upper border moves backward and the lower border forward. But if the connections are unbroken, the movement thus communicated to the manubrium is inhibited not only by the gladiolus, to which it is attached, but also and especially by the clavicles. For since the sternal extremities of the latter project behind and above the articular surfaces on the manubrium, while they are covered by it (? obscure! Hoffmann suggests the manubrium?) in front, the line connecting the acromial with the external extremities of the clavicle runs from above and without, downward, inward, and forward, therefore they are braced firmly against the posterior edge of the manubrium during the inspiratory movement of the entire sternum, and thus assist, by assuming a more horizontal position (obscure again), in moving the upper border of the manubrium forward and away from the vertebral column." Freund's explanation is marred by the obscurity of the language, which is often quite unintelligible and destroys the value of his otherwise painstaking investigations. His fate should be a warning to all those who attempt to give verbal descriptions of complex anatomic relations. I attribute the want of recognition that this author has secured to the fact that he is so hard to read.

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* Seventeenth Congress für Innere Medicin, verhandlg., p. 590.

† The second rib is joined to the manubrium and gladiolus by a double joint, and does not inhibit the movement of the manubrium.

‡ "Der Zusammenhang," etc., p. 18.

In addition, we have a valuable explanation by Landerer in regard to the behavior of the sternum in a healthy individual. The sternum has two movements: (1) an upward movement, which is most pronounced at the upper border of the manubrium because the first costal head is really only adapted for elevation; (2) a forward movement, which is greatest in the lower portion and least at the extreme upper extremity of the bone. The difference in the movements of the various portions of the sternum is made possible by the manubriogladiolar articulation and by the elasticity of the bone. In emphysema the forward movement is always diminished, while the upward movement is performed uniformly by the entire sternum. This corresponds with the results obtained by Rothschild.

In addition to the sternal angle, an anterior curvature of the sternum is observed in many cases similar to the posterior curvature of the vertebral column. The bone presents an anterior convexity, the most prominent point of which corresponds approximately to about the level of the fourth rib; above that point the upper segment is more depressed than the lower segment. In emphysematous subjects the sternum is often straight. The accurate descriptions of the breast-bone in emphysematous subjects of various ages given by Sibson (*l. c.*) cannot, in my belief, be regarded as having a general application. He says that the sternum in youthful individuals is arched, and that the prominence increases from above downward to a point one inch above the xiphoid cartilage, when the prominence suddenly disappears, so that the xiphoid cartilage occupies a depression. At the level of the first to the third ribs he says that the sternum projects beyond the costal cartilages, while the fourth and fifth costal cartilages project in front of the sternum.

In the adult the upper extremity of the sternum is more prominent, especially at the junction of the first and second ribs. Between the cartilages of the fourth pair of ribs, which are very prominent, and especially between those of the fifth pair, the sternum forms a depression. The sixth and the seventh cartilages are but little more prominent than the fifth, whereas these cartilages in youthful individuals are distinctly depressed. The costal angle at the xiphoid process is unusually great.

In senile emphysema the sternum becomes gradually more and more prominent from above downward, but the cartilages of the fourth and fifth ribs do not project, the sternum and the costal cartilages occupying the same plane.

7. The various factors that have been discussed so far explain the interesting **general impression** produced in many cases by an emphysematous patient. The peculiar look and expression of face of an individual who is in constant want of oxygen cannot be described, striking as it is to the observer. The patient looks cyanotic, the eyes are bright and somewhat protruding, the nose, the lips, especially the lower lip, are thickened. The engorgement of the entire venous system is especially marked in the neck and in the upper extremities, where the veins appear as thick blue cords interrupted by nodes that correspond to the positions of the valves. The neck is short and thick; the sternocleidomastoid, scaleni, omohyoid, and trapezius muscles are prominent and appear extremely short, while the intervals between the muscles are marked by deep depressions. The shoulders are drawn up, and the ribs are more horizontal than in the normal subject. The sternum as well as the clavicles are abnormally prominent and rigid, the back is more or less bowed, especially about the middle of the thoracic spine, and the head is carried deep between the shoulders and bent over forward.

In severe cases the patient is found sitting up with his arms on the table, the abdomen swollen, the outlines of the recti muscles distinctly marked. The lower extremities also present dilatation of the veins and often anasarca. The accompanying excellent illustration (Fig. 23), which faithfully reproduces the general appearance of an emphysematous patient, is taken from Rehn.*

Litten's sign is absent and distinct retraction is seen opposite the insertion of the diaphragm on inspiration, unless the thorax has already become too rigid. The sign is therefore most distinct in youthful individuals. But even when the thorax is rigid, the retraction at and below the extremity of the xiphoid cartilage is a striking feature. Stokes'† classic description of emphysema contains some additional features and is well worth reproducing.

> The complexion is commonly somewhat dusky, and the face, while it wears an anxious and melancholy expression, is in many cases bloated, and forms a striking contrast to the condition of the rest of the body. The swelling of the face is probably due to hypertrophy of the cellular tissue and of the respiratory muscles in the face, the former produced by (repeated?) venous congestion, the latter by the excessive exertion of the entire system of inspiratory muscles. The nares are dilated, thickened, and injected. The lower lip is enlarged, the mucous membrane everted and livid, lending the face a peculiar expression of anxiety, melancholy, and disease. The shoulders are thrust forward and the patient habitually assumes a bent position as the result of the repeated tax of orthopnea

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[*] "Die wichtigsten Formveränderungen des menschlichen Brustkorbes," Vienna, Braümüller, 1875.

[†] "Diseases of the Chest," p. 176.

and coughing and because he experiences a certain degree of relief in that attitude. An emphysematous patient is thus frequently found sitting upright in bed with folded hands resting on his knees, and the head bent over forward, the object of this attitude appearing to be to relax the abdominal muscles and to afford a certain support with the arm to those muscles that might interfere with inspiration (?). The shape of the chest is altered to such an extent by the habit of bending over that I have seen numerous cases in which the acromial, intercapsular, and supra- and infra-spinous surfaces were practically horizontal. The point of the scapula under such circumstances becomes unusually prominent; in front the clavicles appear arched and prominent and the triangular spaces that correspond to the insertions of the sternocleidomastoid and scaleni muscles are unusually deep. The cellular and fatty tissue of the neck appears atrophied, but the muscles of inspiration, especially the sternocleidomastoid and scaleni muscles, are hypertrophied and the thyroid cartilage as a rule is prominent and hard, as though ossified. When the chest is examined, other important changes are observed. The sternum has lost its flatness or relative concavity; it is thrust forward and is rigid both in the longitudinal and in the transverse direction. The intercostal spaces are wide, but not distended as in emphysema; the upper half of the chest appears smooth and convex from the hypertrophied condition of the pectoral muscle produced by the constant interference with respiration. When the thorax is examined from the side,

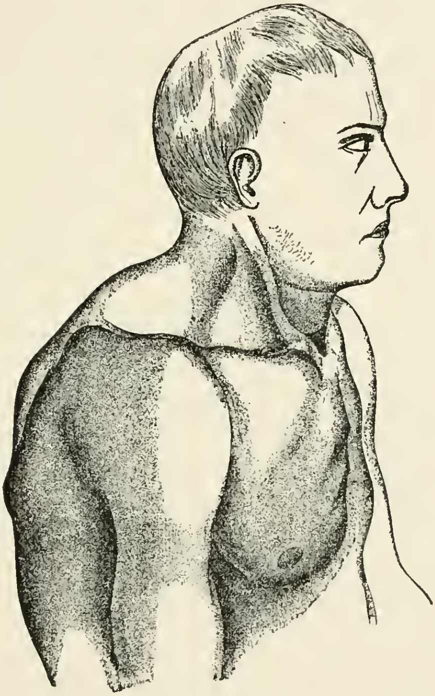

FIG. 23.

however, the intercostal spaces appear exaggerated, and there is no sign of bulging. When the diseases with dilatation of the alveoli are compared with emphysema, the upper portions of the thorax are found to present an appearance of smoothness and dilatation in the former, while a similar appearance is observed in the lower portions in the latter disease. This peculiarity is commented upon and critically discussed by Walshe.

The lateral portions of the chest are deeper than normal and the convexity by no means corresponds to the convexity seen in the anterior and posterior portions of the thorax.

8. On **palpation** the entire thorax, the bones, the cartilages, and the muscles, are found to be extremely hard, the result of the abnormal tension to which they are permanently subjected, as well as of the calcification of the cartilages. The apex-beat cannot be felt, but a distinct thrill is obtained at the lower extremity of the sternum, at the xiphoid process, and in the epigastrium. The thrill is to be attributed to the hypertrophy and downward displacement of the heart and to the congestive enlargement of the liver (compare pp. 332 and 336).

9. On **percussion** the boundaries of the lungs are found to exceed the normal, particularly along the lower border. Occasionally the upper portion of the lung also becomes enlarged, as has been mentioned above; but as a rule the limit of resonance is not affected, at least in quiet respiration, although during violent cough a tumor appears above each clavicle. In percussion downward some difficulty is experienced in finding the cardiac dulness; in many cases only a small indistinct area of dulness is found below the fifth rib, between the sternum and the left mammillary line. When the margins of the lungs are distended, even if the heart is greatly enlarged, there may be no distinct increase in the absolute cardiac dulness. In the severer grades of emphysema the heart is found completely covered by the lung when the thorax is opened, and it is evident that for this condition to be produced the connective tissue between the heart and the sternum must be pushed aside by the pressure of the distending layers of the pleura.

The dome of the diaphragm is lower than normal; the absolute liver dulness begins at the seventh rib in the axillary line; the splenic dulness is small and indistinct, and begins at the tenth rib. Behind, the downward displacement of the diaphragm is less marked. Quite often there is some collection of fluid posteriorly, so that on percussion, with the patient in the erect position, the diaphragm appears to occupy its normal position at the tenth rib. If, however, the patient is asked to bend over forward, the dulness is found to begin at the twelfth rib. Even when the patient presents no other signs of edema, the possibility of the presence of this small amount of exudate is always to be borne in mind.

As a result of the low position of the diaphragm and the accompanying dilatation of the lower aperture of the thorax, the complementary spaces are opened; they diminish in height and become more and more completely filled with pulmonary tissue. The interference with the mobility of the lower border of the lung which is thus produced can be demonstrated by percussion during deep inspiration and expiration; in front, over the liver and stomach, and somewhat less readily over the heart; behind, near the bases.

The lower limit of the pleural sacs at the vertebral column is generally said to be the twelfth rib. At this point the boundary-line is almost horizontal; it even gradually rises and courses about the lateral portion of the thorax, so that on the anterior side of the thorax it intersects the junction of the cartilage with the bone of the seventh rib, and at the right sternal border corresponds to the upper border of the seventh rib, or to the sixth interspace. On the left side the line sometimes appears to be somewhat lower, and of course ends at the pericardium.* In severe grades of emphysema the lungs may extend as far down as the lower border of the pleura just described.

Liebermeister asserts that the pulmonary tissue of emphysematous patients presents a certain dry rigidity which is possibly in part responsible for the diminution in volume and duration of the percussion note.

Biermer's description of the changes in the percussion note is, however, more generally accepted. The note sometimes becomes hyperresonant, and even suggests a tympanitic quality, a sound that Biermer has compared to the resonance of a paper box, and which is therefore known as "box-tone" (*Schachtelton*). This term is too often incorrectly used; many observers invariably hear a box-tone when they have an emphysematous patient before them. As a matter of fact, a clear, well-marked box-tone is a rare phenomenon. Thompson † is quite right when he says that it can only be elicited by light percussion with the finger as a plexor, the finger used as a pleximeter being placed in the intercostal space. Rosenbach,‡ who devotes much of his attention to box-tone, describes it as a very loud (and I should like to add relatively high), but not tympanitic sound—a true parietal resonance, with the production of which the air contained within the hollow space has nothing to do. The true tympanitic note occasionally heard in compensatory emphysema is not to be confounded with box resonance.

10. **Mensuration** of the thorax is usually neglected; nevertheless the method numbers a good many followers, and it is well known that in the examination of recruits, and in life-insurance examinations, it is regularly used. Fröhlich§ prescribes for the examination of recruits, the measurement of the chest circumference in the line passing through the two nipples in front, and im-

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* Pansch, *l. c.*, has collected a number of cases presenting deviations from the normal in this respect.

† *Brit. Med. Jour.*, 1880.

‡ *Deutsches Arch. für klin. Med.*, vol. XVIII.

§ "Die Brustmessung im Dienste der Medicin," Leipzig, 1894.

mediately below the angles of the scapula behind. The arms are held at right angles to the sides of the body. With the man in this position the circumference is to be measured twice, once after inspiration, and once after expiration. The average chest measure of healthy young men, twenty years of age, was thus found to be between 89 and 82 cm., so that the average expansion of the chest is 7 cm. The current assumption that a man, to be fit for military service, must have a chest measurement greater than half the length of the body (in addition to other qualifications) is criticized by Fröhlich. He finds that in men measuring 165 cm. and over, the chest, measured after complete expiration, does not attain half the length of the body. He gives an interesting table (pages 24, 25, *l. c.*) showing the relation between the length of the body and the smallest chest measure. These results, obtained from healthy young individuals, afford a basis for judging the corresponding measurements in emphysematous subjects. As yet there is no satisfactory literature on the subject of mensuration in emphysema from which general conclusions might be drawn; all that is known is that the chest expansion in these subjects is usually much less than 7 cm.

Fröhlich's method of mensuration is for obvious reasons useless in the female sex. Other methods have, it is true, been devised which are better adapted for women, but they have not made their way into general practice. A full description of the various mensurations practised on the thorax will be found in Wintrich's "Handbuch." The more recent studies are found in Riegel's work, "Die Athembewegungen." As nothing of importance either from a practical or a theoretic standpoint has resulted from these investigations, I shall not dwell on them.

11. **Auscultation.**—The *breath sounds* are very weak; in many cases vesicular breathing is altogether absent and in its stead there is heard a distant, ill-defined puff. Expiration is as feeble as inspiration, although it is somewhat prolonged. In many cases bronchiectasis is present, and the sounds heard in the chest are those that belong to that disease. In extreme cases the respiratory murmur has been so weak that nothing at all could be heard and pneumothorax was thought of. It is for this reason that the differential diagnosis between emphysema and pneumothorax is always mentioned in text-books. Tactile fremitus is quite marked, its production being evidently facilitated by the rigidity of the thorax.

The *râles*, of course, depend altogether on the extent and on the stage of the catarrh. The presence of numerous small, sibilant, and sonorous râles, heard both on inspiration and expiration, may be regarded as characteristic of emphysema, although similar sounds are heard in any diffuse bronchitis, and particularly in asthma. The râles are more apt to be dry than moist. Skoda asserts that in cases of emphysema associated with the production of large bullæ, a dry coarse râle, similar to the clacking sound made with the tongue, is heard at the end of inspiration, either preceded by ordinary râles or unaccompanied by any other adventitious sounds.

12. Although the third fundamental symptom of emphysema—**catarrh**—has always engaged the attention of clinicians, it has never been exhaustively studied. The varieties and changes in the condition are too great. There must be two kinds of catarrh. The first is the *causal catarrh*. This, in my opinion, may be a simple bronchitis, or it may be an asthmatic form of catarrh, or a form that develops in various kinds of stenoses in the large air-passages, the larynx, the nose, and the trachea. These forms alone, and the variations which they present in the course of years, would make up a sufficiently kaleidoscopic clinical picture. To these in the course of time *congestive catarrh* is regularly superadded by the slowing of the blood-current incident to the increasing cardiac weakness and insufficient aspirating power of the lungs.

According to Eppinger, congestive bronchial catarrh affects the bronchi of the first, second, and third orders, and is accompanied by very little swelling of the mucous membrane, which, however, is deeply injected and produces a watery secretion. The causative catarrh, on the other hand, affects the small bronchi and is accompanied by the production of a glairy, extremely tenacious secretion. In a number of cases, however, developing gradually in patients suffering from heart disease, the congestive catarrh itself becomes the cause of emphysema.

Hence there are undoubtedly cases in which the distinction is not clear, or does not exist at all. Ordinarily the catarrh accompanying emphysema is associated with mucoid, more or less tenacious sputum which always contains an abundance of fluid and a small number of morphologic elements, especially pus cells. If large masses of pus cells are found, the condition is not a simple emphysema catarrh, but must be regarded as a complication. The catarrh of emphysema, as has been said, is mucous; the sputum may be, in fact, defined as frothy from the presence of masses of small and extremely small air-bubbles. This frothy sputum is not peculiar to asthma, but occurs in any simple catarrh accompanying an advanced stage of emphysema, for it is very often found to be quite free from eosinophile cells. The peculiar nature of the sputum is evidently due to the fact that the mixture of mucus and air begins in the smallest bronchi.

Laennec attributed especial importance to the so-called dry catarrh (*catarrhe sec*); that is, a catarrh associated with very scanty and exceedingly tenacious sputum which is laboriously detached from the walls of the bronchi after severe coughing. There is no sharp distinction between mucous, frothy catarrh and dry catarrh; but when the latter is extensive, it becomes exceedingly distressing to the patient on account of the severe paroxysms of cough which are evidently calculated to bring about a rapid aggravation of the emphysematous condition in the lungs (see also p. 281). On staining the sputum with eosin it is found, in a certain number of cases, that the catarrh is distinctly eosinophile. As a rule, this is the case in asthmatic patients, who, as we know, furnish such a large contingent in emphysema. But even among these patients with eosinophile catarrh a certain number are found who have never had true asthmatic attacks and do not present any other signs of asthma. It may be assumed that the asthma in these patients represents a rudimentary form; but, to avoid dealing too extensively in mere hypotheses, one will be forced to assume the presence of a peculiar eosinophile catarrh, the causes of which, like the significance of eosinophilia in general, must be still considered doubtful.

In all other emphysematous patients the cells in the sputum simply take on a uniformly weak stain with eosin, and the method yields no special results. If a number of specimens of sputum are stained with eosin, two other distinct forms of cells will be found; one of these varieties takes on a very complex stain, the cells containing deeply stained, irregular, fragmentary nuclei surrounded by a broad areola—the whole embedded in a pale red ground-substance. These cells evidently represent polynuclear leucocytes that have entered the air-passages from the vessels and have undergone changes in the secretion of the mucous membrane. The cells of the other variety are characterized by the presence of vacuoles which, under certain circumstances, may become so large as to occupy the entire cell, leaving only small remains of the cell contents in the form of deeply stained lines and dots in the center of a bright area surrounded by a well-defined membrane. I formerly described these cells as "tubes" (Schläuche, tube casts) taking them to be derivatives of epithelial cells. But it appears that morphologic elements similar in every respect to these tubes may be produced by a profound alteration of the pus-corpuscles. If these two varieties of cells are found in the sputum, it may be concluded with certainty that the existing catarrh has nothing to do with emphysema, and has already become aggravated by some complication, or exists quite independently of the emphysema.

In a certain proportion of cases I have observed amylaceous bodies in the sputum of emphysematous subjects; in others I have found crystalline structures which, on the addition of acid, dissolved with the liberation of small bubbles of gas, showing that they probably consisted of calcium carbonate. These crystals are not constant and are also found in many other forms of catarrh of the respiratory passages.

The *cough* of emphysema also deserves a few lines, as it plays a considerable part in the development of the disease. It is often extremely distressing, laborious, and dry, and always affects the general condition of the patient unfavorably. The dry catarrh which Laennec considered especially dangerous is chiefly characterized by the presence of this cough. As long as a fairly copious amount of mucus is brought up without great exertion, the patients are comparatively comfortable. The cough is therefore more or less intimately connected with the nature of the catarrh, but there are also cases in which cough occurs as an independent trouble, and in which catarrh is practically absent, although the cough almost drives the patient to distraction. Certain neurotic factors are probably in part responsible for such a condition, but as yet nothing definite is known about their influence.

In a certain proportion of the cases blood is found in the sputum. They are usually cases associated with congestion, in which the right heart has suffered severely. Numerous heart-lesion cells are then found in emphysematous sputum. Occasionally a small streak of blood is observed in the mucous expectoration following a violent paroxysm of cough, or a few unchanged red blood-corpuscles may be demonstrated microscopically in the sputum. Is there such a thing as true hemoptysis irrespective of a congestive base? Is it possible for bright frothy blood, practically unmixed with sputum, to be expelled in large masses? Most authors are agreed that hemoptysis occurs as a rare symptom in emphysema. If cases with distinct congestive phenomena are excluded,* those that remain appear to me very doubtful, and I cannot help suspecting the presence of phthisis. At any rate, the trustworthy observations are so few that I am inclined to doubt the occurrence of hemoptysis (see Complications).

There is one form of pulmonary hemorrhage which might be regarded by many as true emphysematous hemoptysis, and must therefore be mentioned. I refer to the hemoptysis described by Andrew Clark.† It occurred in elderly, well-nourished individuals with well-marked arteriosclerosis and emphysema. From the history, the presence of an acute or rheumatic diathesis with a tendency to cutaneous eruption and bronchial catarrh was obtained. The hemorrhage in several cases was severe enough to threaten life, or even fatal, and the use of calomel and saline laxatives was followed by most satisfactory therapeutic results. In the fatal cases Clark could find no explanation for the hemorrhage macroscopically, as the only prominent symptom was emphysema. Microscopically, however, he found numerous small pulmonary vessels in a state of degeneration, and these had produced the lacerations and multiple hemorrhages. He calls this hemoptysis arthritic, and, disregarding the importance of the emphysema, believes that the arterial degeneration and its consequences were due to the peculiar constitution and improper mode of life of the patients. It is quite evident that on this theory the hemorrhages were not cases of emphysematous hemoptysis at all. I have not been able to find any more recent investigations on similar occurrences in the literature. Huchard ‡ some years ago reported cases of periodic pulmonary hemorrhages depending on arthritic disease. As similar conditions are mentioned in alcoholism, this source of error must also be borne in mind. I have so far been unable to find any well-observed case in the literature.

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* Hauff's case, *Württ. Corr.-Bl.*, 1869, "Schmidt's Jahrbücher," 163, p. 289, presented distinct signs of congestion.

† *Lancet*, 1889, II, p. 840. Also Hertz's case, *l. c.*, p. 386.

‡ *Union méd.*, 1883; Ref. *Klin. Centralblatt*, p. 743.

I have myself seen several cases of hemoptysis in which it was impossible to determine the cause and in which I could exclude with absolute certainty phthisis, bronchiectasis, and any other process capable of producing severe alterations in the pulmonary tissue. The possibility of cardiac lesion could also be excluded. In two of the cases the patient's general habit was such as to suggest emphysema, but in both cases the hemorrhage was unattended by any untoward results and remained unexplained. It is probable that every busy practitioner comes across at least one inexplicable case of hemoptysis, and it is possible that such cases are occasionally attributed to emphysema; but that, of course, is merely an unfounded assumption.

13. **The Heart.**—The sounds of the heart as well as of the large vessels are weak, because they are muffled, as it were, by the distention of the edges of the lungs. The heart sounds are sometimes better heard over the left lobe of the liver than at the point where the apex must be assumed to be. The sounds, as a rule, are pure; but a more or less distinct, systolic, blowing murmur is not infrequently heard at the apex, although at the autopsy the valves are found to be perfectly sound. Gerhardt is of the opinion that this is due to the obliteration of numerous pulmonary capillaries; in other words, that it is an anemic murmur. Biermer believes that the phenomenon is to be attributed chiefly to the relative insufficiency of the tricuspid valve. As the right ventricle regularly hypertrophies and in time becomes dilated and undergoes fatty degeneration, the development of relative tricuspid insufficiency is quite probable, and if the sound is heard best at the lower end of the sternum, it is another sign in favor of that supposition. But how does the case stand when it is heard much better in the region of the apex?—for that is not an uncommon occurrence. It may be anemic, but I am of the opinion that it is usually connected with changes in the cardiac muscle. It is quite certain at least that mitral insufficiency may be very difficult to diagnose in pulmonary emphysema. It is even asserted that accidental diastolic sounds have been heard over the apex in emphysematous patients. Ganghofner,* in his work on "Dilatation of the Heart," page 35, cites a case of severe emphysema in which a loud systolic murmur was heard in the tricuspid area, and both a systolic and a diastolic murmur over the mitral valve. At the autopsy the left pulmonary orifice admitted three, and the right four, fingers; all the valves were normal, the walls of the heart were thickened, and the muscle was yellowish and brittle. Finally, it is to be mentioned that a double diastolic sound may be heard at the apex, the

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* *Prager Vierteljahrsschr.*, vol. cxxx.

second of the two occurring immediately before the systolic sound (presystolic sound).*

It is assumed as a matter of course, from the low position of the diaphragm, that the heart also occupies a lower position; but this statement is not to be accepted offhand. Bamberger† many years ago gave a better explanation of the true condition of affairs. He said: "As the arch of the diaphragm diminishes and takes up a lower position, the base of the heart, which it supports, must be correspondingly depressed and must approach the epigastrium, from which, under normal conditions, the right border of the heart is separated by the distance equivalent to, or slightly exceeding, the entire length of the xiphoid cartilage. But the same is not true of the apex, for the latter does not rest upon the dome of the diaphragm, but upon the ascending crura. Hence, although the base of the heart is lowered, the position of the apex remains the same, unless the flattening of the dome of the diaphragm is so great that the vertical direction of the ascending crura is altered, in which case the apex becomes slightly depressed."

On the authority of Kiwisch,‡ it is always stated that the apex-beat during deep inspiration descends into the next intercostal space, and performs a corresponding upward movement during deep expiration, but I believe this statement is erroneous. It is distinctly contradicted by the Röntgen-ray picture, and it seems to me a hazardous undertaking to draw conclusions from the result of palpating a thorax engaged in active respiratory movements. There is too much danger of fallacy from suggestion. The appearance in the Röntgen-ray picture convinces me that a normal heart is relatively independent of the centrum tendineum, for during deep inspiration a clear interval could even be seen below the heart. The heart is not in close contact with the centrum tendineum, although it may under ordinary circumstances derive some support from that structure. During normal, quiet respiration the centrum tendineum does not move at all. The depression which it suffers through the diaphragm is compensated by the corresponding elevation of the thorax, in which the centrum tendineum participates. In forced inspiration the shadow of the diaphragm becomes distinctly separated from that of the heart.

But this separation is never seen in emphysematous subjects, in whom the heart shadow and the diaphragmatic shadow, or, better, the liver shadow, are intimately united; and since in these patients the diaphragm, with the centrum tendineum, occupies a somewhat lower position with reference to the ribs, the heart must also be displaced downward. Bamberger accordingly found that a needle inserted into the epigastrium penetrated the right ventricle near the auricle, but the value of this experiment must not be overestimated.

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* Traube, "Gesammelte Abhandlungen," III, p. 12.

† *Würzburger med. Zeitschr.*, I, 1860, p. 221.

‡ *Prager Vierteljahrsschr.*, vol. IX, p. 153.

Although I agree in the main with Gerhardt, I differ with him in certain points. He says: "In an emphysematous subject the heart is displaced downward, and although the apex-beat is seen approximately at the same place, a portion of the heart—namely, the border of the right ventricle—becomes sufficiently depressed and occupies such a position as to produce a pulsation that is both visible and palpable below the xiphoid cartilage." Since this statement was made, the pulsation in the epigastrium has always been attributed to the right ventricle. But this is far from being true in every instance. If the experiment of inserting a needle in the epigastrium, after Bamberger, is performed repeatedly, a number of cases will be found in which, although during life there was a distinctly marked epigastric pulsation, the needle does not enter the heart at all, but pierces the liver. Many of these epigastric pulsations are therefore simply due to the vibrations of the liver transmitted from the heart. In other cases the pulsation is to be attributed to the abdominal aorta, for there is a distinct interval between it and the apex-beat. Quite frequently the pulsation is distinctly diastolic, and then it is evident that the pulsation begins in distention of the right ventricle and is transmitted either through the abdominal walls or to the liver. The possibility is also to be borne in mind that the heart contracts during systole, so that a depression is produced where before there was a distinct bulging. Hence the systolic retraction of the intercostal spaces often misleads the observer and suggests the thought of pericardial adhesion, when, as a matter of fact, there is no sign of any such condition. But it is evidently quite possible for this systolic depression to occur in the epigastrium if the heart is displaced somewhat downward, and the logical consequence of such a depression is a diastolic elevation. To assert in a given case that the heart has become displaced into the epigastrium is, in my opinion, possible only when the abdominal walls are so flaccid and attenuated, as sometimes happens, that the heart is plainly felt in contact with the liver, a groove being clearly made out in which the finger can be inserted between the heart and the liver. If the heart is displaced as far as the epigastrium, the disease must be unusually severe, whereas epigastric pulsation is quite a common symptom. It is also to be borne in mind that the insertions of the diaphragm at the xiphoid cartilage and at the seventh costal cartilage necessarily interfere with downward displacement of the heart, even if the heart were simply supported on the diaphragm and had to follow its movements slavishly, according to a view that is sometimes expressed. On the contrary, the heart finds sufficient support in its blood-vessels and in the pericardium, which has numerous attachments on the sternum and on the posterior mediastinum; and even though it is adherent to the diaphragm, it is not forced to follow its movements as a whole. That the centrum tendineum under all circumstances is less movable than the lateral portions of the diaphragm is shown by the well-authenticated fact* that the displacement of the liver in deep inspiration and expiration appears slighter as it approaches the median line, and greater as it moves nearer the axillary line. The apex-beat is often invisible, because the region of the apex is covered by lung.

Although the enlargement regularly affects the right heart, cases in which enlargement of the left heart is mentioned are found in the literature.† These cases are in need of further explanation. Traube, who also observed dilatation and hypertrophy of the left heart in association with similar conditions in the right ventricle, attributes the condition to repeated and persistent contractions produced in numerous smaller branches of the aortic system by the accumulation of CO_2. He calls attention to the fact that in a great variety of diseases of the respiratory apparatus the appearance of severe dyspnea and cyanosis is accompanied by a material increase in the tension of the radial arteries.‡

14. The **circulation** is subject to a number of injurious influences in emphysema. The most important of these are the feebleness of the respiration, the diminution in the movements of the thorax (the pressure of the incarcerated air during the frequent paroxysms of cough), and the diminished elasticity of the pulmonary tissue. In comparison with these conditions the destruction of numerous capillaries is probably of very small significance (compare p. 261). The immediate consequences of these injuries consist in a greater amount of work being thrown on the right ventricle, which accordingly becomes regularly hypertrophied in this disease. As long as the hypertrophy suffices to fulfil the demands of the organism we may speak of compensation in emphysema, and this compensation or compensated emphysema may last indefinitely, without undergoing a change for the worse, if the general conditions of life are favorable. As a rule, however, compensation in time becomes inadequate; more and more blood stagnates in the right heart and in the auricle; the cavities fail to empty themselves completely, and become dilated. From this condition to degeneration of the thickened muscle is but a step, particularly as alterations in the coronary vessels and in the heart always appear to be present, and to these I should like to direct the attention of the pathologists.

This hypertrophy and dilatation of the heart can never be determined by ordinary methods of examination, because percussion is attended with so many difficulties. But before long the stasis spreads to the large vessels of the neck and to the liver, and thus becomes unmistakable. The congestive phenomena may be quite as marked as in pronounced cases of mitral disease. The excessive distention of the large veins in the neck, in conjunction with the cyanosis, produces an exactly similar impression. In fact, pulsation is observed in the veins of the neck in the higher grades of emphysema just as in mitral disease, either because of true insufficiency of the tricuspid valve following degeneration of the hypertrophied ventricle, or because, even without tricuspid insufficiency, the pulmonary valve alone has become insufficient on account of the excessive congestion, so that the contraction of the right auricle drives the blood back into the jugular veins.

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* Gerhardt, "Diaphragma," p. 41.

† Duhamel, *Gazette hebdomadaire*, 1865, No. 38, p. 600.

‡ "Gesammelte Abhandlungen," III, p. 4, 124.

As the congestive symptoms increase in severity, the pulse becomes small and weak, and, as a rule, distinctly accelerated.

15. **Liver.**—At first the liver is unaffected in emphysema. Gradually, however, it becomes displaced downward along with the descent of the dome of the diaphragm. The lower border is then distinctly felt, and the viscus appears to be enlarged, although it really is not. Gradually, however, as the congestion and cardiac insufficiency increase, it becomes larger and harder and approaches the condition characteristic of the last stage of mitral disease, as the circulatory disturbance in the lesser circulation and the resulting congestion must be exactly alike in the two cases.

The greater the depression of the diaphragm, the more completely is the liver freed from its covering of lung. The relative liver dulness diminishes by a progressive descent of its upper limit, while the absolute liver dulness increases. The movements of the lower border of the liver during respiration of course progressively diminish with the diminution in the movements of the greatly depressed diaphragm.

The liver in time becomes more and more enlarged and congested, and can be more and more readily felt. The amount of movement in the diaphragm can be determined by observing the movements of the lower border of the liver.

16. **Spleen.**—The spleen in emphysematous patients presents no special symptoms. It must, of course, be displaced downward along with the liver and the diaphragm, but I have never been able to palpate it in these patients, and all that is found on percussion is that the dulness is indistinct and difficult to determine. Splenic tumor forms no part of the symptom-complex of emphysema.

17. **Stomach and Intestinal Canal.**—Emphysematous patients usually have a very good appetite; even when the severity of the cough is extreme and the sputum exceedingly tenacious, there is no tendency to vomit.

They often complain of a certain degree of constipation, which is not difficult to understand in view of their limited ability to indulge in exercise. After the dyspnea has attained a certain degree, the distention of the stomach incident to a copious meal is more or less troublesome, and emphysematous patients are often inclined to attribute their disease to stomach trouble. In advanced cases of venous stasis some authors mention congestion and thickening of the gastric mucous membrane.

18. **Urinary Organs.**—The kidneys of emphysematous patients in severe degrees of the disease are subject to the same conditions of stasis as the liver. The urine presents all the signs of genuine congestion. In addition, there are alterations dependent on insufficient oxygenation of the tissues, the significance of which awaits further study with the aid of theoretic investigations (see Embolism).

19. **Sexual Apparatus.**—No special pathologic conditions in these organs have been observed in emphysema.

20. **Larynx and Upper Air-passages.**—The connection between diseases of the lung and disease of the external air-passages, by which is meant the pathway from the entrance of the nose to the vocal cords, has excited considerable interest in recent times. The question has already been adverted to in the section on Etiology. We have here to deal with the conditions that develop after the emphysema, and thus become symptoms of the disease. There is no doubt that emphysematous patients present a marked tendency to laryngitis and pharyngitis—a tendency that, as a rule, is due to the congestion, which induces a chronic hyperemia, and is in turn aggravated by the laborious expectoration of the tenacious sputa.*

21. **Nervous System.**—Many emphysematous patients complain of all kinds of "rheumatic" pains; or, at all events, if asked whether they suffer from rheumatism, eagerly answer in the affirmative. But many of these people are addicted to alcohol and present characteristic forms of alcoholic neuralgia. Since the laws in regard to disability were framed, and emphysema is regarded as a possible consequence of traumatism—and I should like to ask what under the canopy is not a possible consequence of traumatism?—I have seen a number of emphysematous patients who some time or other had received a blow somewhere about the head and had become emphysematous in consequence. One patient had such violent pains at the site of the alleged traumatism that he became a confirmed morphin-eater. He was a heavy drinker, but the possibility that the blow might have had something to do with the pain and the emphysema could not be absolutely denied, and he received a full annuity. I have had occasion to observe pain of this kind several times in emphysematous individuals; they were all cases of alcoholic neurosis. But your genuine emphysematous patient has no pains that can be regarded as symptoms or results of his disease. When such a statement is made, the case should be carefully examined. As a rule the pain is referred to the back or to the sides, and is evidently con-

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* Friedrich, "Rhinology, Laryngology and Otology," W. B. Saunders & Co., Philada., 1900, p. 31.

nected with the cough, as it increases and diminishes along with the improvement and exacerbation of that symptom.

22. **The Extremities.**—No special indication to devote attention to the legs has so far been brought forward. They are apt to get cold, and the knees become blue as in all cases of cyanosis. During the stage of cardiac weakness they of course become edematous. Acrocyanosis is often very conspicuous in the nose. The hands of emphysematous patients are not, of course, deeply cyanosed, because these patients are less cyanotic than individuals suffering from grave cardiac disease. On the contrary, marked anemia often develops toward the end, so that cyanosis is rarely very great. It is probably for this reason that an emphysematous patient, in spite of his congestive symptoms, rarely has clubbed fingers like other patients with engorgement of the right heart and severe cyanosis, particularly cases of pulmonary stenosis, in which the engorgement is regarded as the cause. Clubbing of the fingers is frequently observed in bronchiectasis and in phthisis, whereas in emphysema it is so rare that it is not, as a rule, included among the symptoms of the disease. According to Waters, the temperature of the skin is always lower than normal.

23. **Metabolism.**—The general health of the patient is not impaired except in the higher grades of emphysema. During the initial stages it is not affected, and one sees plenty of obese sufferers from the disease. In them emphysema is as a rule overlooked, because the dyspnea appears to be sufficiently explained by the obesity, and the emphysematous appearance of the thorax by the enlargement of the abdomen. There is no doubt, also, that in such patients it is much more important to recognize and treat the obesity than the emphysema.

In advanced stages of the disease, on the other hand, the patients are always more or less cachectic, as nutrition is evidently impaired by the insufficient ventilation of the lungs. Later the bad effect of the general congestion in the organs makes itself felt in the digestive apparatus. Young patients are more cyanotic, while elderly ones incline toward anemia as senility develops.

One might be inclined to utilize the results obtained in studying the effects of dyspnea on metabolism in the study of emphysema. In some experiments an insufficiency of oxygen was induced by having the subject breathe in rarefied air,* and an increased decomposition of albumin, especially in the form of increased excretion of uric acid through the urine, was determined. Lepine,† by introducing a mechanical obstruction to the entrance of air, brought about a marked increase in the percentage of carbon dioxid in the expired air, while the absolute elimination of carbon dioxid was diminished. The increase in the decomposition of albumin determined by Fraenkel, Zuntz and Oppenheim, and Senator was denied by Eichhorst,* and again confirmed by Penzoldt and Fleischer.† Since the dyspnea is always accompanied by increased muscular activity, the latter unquestionably plays an important rôle in its production. A similar increase in the decomposition of albumin is demonstrated in the work of Reale and Boesi,‡ whereas Colasanti and Palimanti § determined an absolute and relative diminution in the excretion of urea and nitrogen when the respiration was mechanically obstructed. The results of these investigations are not directly applicable to emphysema, and therefore have only a theoretic interest in this connection.

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* Fraenkel, 1876, 1880; Levy, 1882.

† "Jahresbericht über die Fortschritte der Thierchemie," 1882.

COMPLICATIONS.

During the stage of emphysema a number of important complications may make their appearance. Either the causative **bronchitis** or the causative **asthma** may reassert itself. This is, in fact, quite a common occurrence, and really belongs to the clinical picture of the disease. The emphysematous subject, on his own showing, is especially predisposed to catarrh in winter, and to have an attack of asthma when he is exposed to heat, to dust, or to unfavorable atmospheric conditions for which he has an idiosyncrasy. Then, again, asthma and bronchitis may coexist, so that an asthmatic patient may have a simple bronchitis which has nothing directly to do with his asthmatic attacks, and a bronchiectatic patient may have attacks of dyspnea which may resemble the asthmatic attacks as closely as one egg resembles another, and the only possible way of making a differential diagnosis that I can think of is to examine the sputum for eosinophile cells.

Pneumothorax is produced by the laceration of an alveolus situated underneath the pleura. The condition is usually benign and ends in recovery. The result, however, evidently depends on the character of the air within the cavity produced and on the nature of the opening. If the air is polluted by an excessive number of micro-organisms, an inflammation may develop; and if there happens to be an infiltration at the site of the opening, it may close up with difficulty or show no tendency whatever to heal. Absorption of the air then becomes impossible, as fresh air constantly takes the place of the air absorbed whenever the lung is subjected to any degree of pressure. A favorable prognosis in emphysematous pneumothorax is based chiefly on the fact that the air becomes absorbed during the first twenty-four hours and that the lung is capable of expanding. The ruptured alveolus is then collapsed and the opening obliterated. If the lung expands again, there is every probability that the opening will remain closed, because the pressure on the opening is very slight, and the distended alveoli in the neighborhood, which are not very elastic, readily fill out and occupy the space.

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* "Jahresbericht," 1877.

† *Virchow's Archiv*, vol. LXXXVII.

‡ "Jahresbericht," 1894.

§ *Ibidem.*

Interstitial emphysema is produced by severe coughing. As long as it is confined to the lungs it cannot be recognized, although Laennec regarded a dry crepitant râle with large bubbles as characteristic enough to justify a diagnosis of interstitial emphysema. This view has, however, been abandoned; and we now believe interstitial emphysema can be recognized only when subcutaneous emphysema begins to declare itself in the neck by the characteristic crepitation transmitted to the palpating finger. Opinions vary in regard to the danger of this condition. Roger states that 17 of his 21 cases were fatal. When it is remembered how many cases are never diagnosed, and that only the most pronounced examples are recognized, and that, moreover, a number of these recover, it is probably a mistake to rate the dangers of interstitial emphysema too high. The patients that I have seen die with interstitial emphysema were in such a condition that they could not in any event have been kept alive for any length of time.

Cardiac complications, over and above compensatory hypertrophy of the right heart, are often observed. Lebert * claims to have found valvular lesions in 10% of the cases (in 8 insufficiency and retraction of the mitral valve, in 2 aortic disease, in 1 complicated lesions; see also page 277). Thrombus formation in the right auricle, or rarely at some other point,—in the vestibule or in the ventricle,—can hardly be called a complication; it is to be regarded rather as a very common terminal manifestation in the normal development of the cardiac hypertrophy and dilatation incident to emphysema.

Collections of fluid in all the cavities of the body are the natural result of the cardiac disturbance, and need not be mentioned as a special complication.

Bronchiectasis is mentioned by many authors. It assumes the regular cylindric form and produces no clinical symptoms.

Gastric disturbances manifest themselves in connection with the venous engorgement; *dilatation of the stomach* has even been described as a final result.

Phthisis may complicate diffuse emphysema, although the association is not very common. The demonstration of tubercle bacilli in many patients previously regarded as emphysematous occasionally comes on the physician as a surprise, and explains many curious complaints, in some cases even febrile movements and loss of flesh, for even pronounced pulmonary changes characteristic of phthisis may be masked for some time by the presence of the emphysema. Accordingly this form has often been included among the so-called latent forms of phthisis. It is said to be characterized by a slow course and limited extent of the pulmonary lesions.

Cerebral Hemorrhage.—Emphysematous patients often complain of headache and a feeling of pain in the head, symptoms that are unquestionably to be referred to the congestion in the venous circulation. The occurrence of *apoplexy* in association with emphysema is probably very exceptional. The arteriosclerosis which favors the development of emphysema is probably also responsible for the apoplexy, when the latter occurs.

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* "Klinik der Brustkrankheiten," I, p. 377.

As many emphysematous patients go through a stage of congestion it is not to be wondered at that any variety of **hemorrhage** may appear as a complication of the disease. The hemorrhage most commonly takes the form of *epistaxis* or *hemorrhoids*, but *hemoptysis, hematemesis, intestinal hemorrhages*, and *hematuria* have also occurred in rare cases. The patients often feel much relieved after these hemorrhages, which may in a sense be regarded as curative efforts on the part of nature. Hemorrhages into the *retina* also no doubt occur as symptoms of congestion. But the case reported by Litten * cannot be attributed to retinal hemorrhages alone.

The **thromboses** that develop in the terminal stage of emphysema are readily explained by the dilatation and weakness of the right ventricle. The right auricle is an especially favorite seat of thrombus formation, and clots are also found in the jugular, the saphenous, and the crural veins, in the vena cava, and in the sinuses of the brain. From the right auricle, and sometimes from the other veins referred to, the thrombus may be carried into the pulmonary circulation and lead to **embolism** of smaller or sometimes even larger branches of the pulmonary artery, with the logical consequences that need not be especially discussed.

The association of **gout** and emphysema must be regarded as quite frequent, since Greenhow distinctly mentions gouty constitution as the chief cause of the development of emphysema. Other authors do not say much about it; but Norman Moore † makes the statement that emphysema is as common as interstitial nephritis in gout. Since bronchitis is regarded by Englishmen as a frequent gouty disease, the emphysema is readily explained in the same way. On the whole, I am of the opinion that alcoholism plays a considerable rôle, and I am inclined to leave the question whether gout may be mentioned as a direct cause of emphysema open to debate.

Obesity is also to be regarded as a not infrequent occurrence in emphysema, and it may quite possibly possess some diagnostic significance by bringing about a tendency to bronchiectasis and cardiac weakness.

Mention is occasionally made of the frequency of **rheumatism** in emphysematous subjects, but Lebert was unable to confirm this assertion in his 860 cases. Greenhow also claims a certain relationship between **psoriasis** and emphysema.

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* *Charité-Annalen*, 1876, 172.

† *Trans. Path. Soc.*, vol. XXXIII.

PROGNOSIS.

The prognosis is unfavorable, inasmuch as there is little prospect of obtaining a complete cure except in a few cases of youthful individuals when the disease is of recent origin and has not yet had time to involve a large portion of the lungs. It is favorable, however, in the sense that life is not threatened for some time, and with due care and favorable external circumstances the patient may live to extreme old age. Still, there is a good deal in the way of treatment that can be done to alleviate the patient's discomfort. If the heart is sound, the prognosis is at once rendered much more favorable. Much depends on the degree of emphysema present. This can be determined by means of the spirometer. Waldenburg asserts that the prospect is unfavorable if the vital capacity, as shown by the spirometer, is reduced one-half or even more; hence the saying: "The ear makes the diagnosis, but the spirometer makes the prognosis."* Much, however, can be accomplished by exercise, and I should not be inclined to regard the spirometric result as unfavorable until every effort to secure a better one had failed.

DIAGNOSIS.

The diagnosis of diffuse emphysema presents no difficulties. The shape of the thorax, the boundaries of the resonant area in the lungs, and the weakened vesicular breath-sounds are considered as the three classic objective signs. Emphysema, it has been said, may be mistaken for pneumothorax, but this can be possible only in cases in which the phenomena of pneumothorax, owing to the presence of adhesions, fail to make their appearance abruptly, so that the condition develops comparatively slowly and never reaches a high degree. If metallic breathing and loss of vocal fremitus are demonstrable, the error will be readily avoided; but when these signs are absent, the mistake has been occasionally made, else the differential diagnosis would not be described so extensively in all the text-books.† In phthisis, pulmonary abscess, and pulmonary gangrene the possibility of emphysema will be borne in mind.

Emphysema may also be confounded with compression of the trachea or of the main bronchi. The question whether pneumonomycosis simulating emphysema exists is still under debate.‡

Except during an asthmatic attack, emphysema will scarcely be confounded with the stage of asthma in which emphysema has not yet developed. When emphysema was still sharply separated from asthma as a distinct disease, the differential diagnosis between the two conditions was much discussed. Dilatation of the thorax is absent and retraction of the lower portion of the thorax is present in asthma.

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* Lermoyez, *France médicale*, 1891, p. 641.

† Homolle, in "Nouveau Dictionnaire," Paris, 1880; Rilliet et Barthez, and Riegel report cases of this kind.

‡ *Lancet*, 1894, vol. L, p. 1252.

Hypertrophy of the lungs is also mentioned in the text-books, but experience shows that symptoms of emphysema do not occur in this condition. The vesicular breathing is not impaired, the respiratory and vital capacities are normal, although the boundaries of the lungs are enlarged. In the rare cases so far reported the conditions were quite abnormal, and were never understood before the autopsy.

The diagnosis of emphysema having once been established, it remains to decide whether the case is one of acute emphysema, as in whooping-cough and in capillary bronchitis, for example, or a case of chronic emphysema.

During the cardiac stage it is sometimes difficult to determine the origin of the condition, as cardiac disease alone or a combination of renal with cardiac disease may simulate emphysema. Errors will, however, usually be avoided unless the clinical picture is complicated by the presence of pulmonary catarrh. As long as this complication persists it will be impossible to arrive at any definite decision.

It is necessary to distinguish between asthmatic and bronchitic emphysema.

Vicarious emphysema occasionally masks other changes; thus, phthisis is often concealed by the condition and leads to the error of diagnosticating a primary emphysema. When emphysema develops in the case of individuals who have formerly suffered from disease of the lungs and show areas of dulness, retraction of the lung, changes in the shape of the thorax, and a general deviation from the characteristic emphysematous habit, the correct diagnosis will usually be suggested by the depression of the diaphragm, by a diminution in its excursions, and by the displacement of the heart. [Such conditions are more readily determined by a fluoroscopic examination. —Ed.]

An emphysematous subject may develop tuberculosis secondarily, and there is no reason to be astonished at the apparent parodox of an emphysematous thorax and the finding of tubercle bacilli in the sputum.

Whether emphysema may coexist with a valvular lesion is often difficult to decide, since the picture of mitral insufficiency is often directly produced by emphysema. The question will have to be determined by percussion. If the two conditions are present together, a correct diagnosis may be obtained through the history.

In children the diagnosis of emphysema is often extremely difficult, because the characteristic barrel-shape of the thorax does not develop, especially in the earlier years of childhood. The furrow of peripneumonic retraction may be produced by any form of dyspnea and is not in the least characteristic of emphysema. A more trustworthy sign is the expiratory distention of the upper portion of the lungs during violent paroxysms of coughing. This sign is often very conspicuous, and was long ago pointed out by Fürst. Those who pretend to distinguish alveolar ectasia, "*volumen pulmonum auctum,*" from "genuine" emphysema, and claim that the term emphysema should be used only in the presence of profound pathologic changes in the tissues, will find great difficulty in making a differential diagnosis in the case of children.

Niemeyer attempts to draw a sharp line of distinction between permanent inspiratory distention of the pulmonary alveoli and vesicular emphysema. This author asserts that in the former case the pulmonary alveoli maintain the degree of distention which they normally possess at the height of inspiration, while in emphysema, on the other hand, the distention exceeds the normal, "two very different conditions, and it seems very strange that they have so often been united in the same description." But it seems to me that this is only natural, for how is it possible to recognize this permanent distention? If the condition has developed rapidly and there is prospect of recovery, one may speak of an acute pulmonary distention; but there is no such thing as a permanent respiratory distention—either the distention returns to normal or it goes on to the development of genuine chronic emphysema. The clinician cannot allow his diagnosis of emphysema to hinge on symptoms that develop only in an advanced stage of the disease, or even require an autopsy for their full recognition. I shall leave it to the pathologists to decide whether Niemeyer's division is to be retained for their purposes.

It is true that there are cases in which the term emphysema is incorrectly applied. In the case reported by Hecker,* in which labor was terminated without artificial intervention after twenty-four hours of labor pains, eighteen hours after the rupture of the membranes, and one hour after the heart sounds had ceased to be heard, and a still-born child was born presenting distention of the lungs and even laceration of the alveoli, it is right enough to say that the case was not one of disease, so much as of a suddenly produced condition—an accident, as it were. In a similar way excessive dyspnea during the agonal period may lead to a distention of the lungs which is designated by many authors as an acute emphysema. It is for this reason that I have said that for practical reasons the term emphysema should be used only when the distention of the lungs has continued for at least eight days, this period having been chosen as sufficient for the development of an actual morbid condition (see p. 293). If the condition lasts less than a week, I prefer the term acute pulmonary distention. In the great diversity of opinions, however, the boundary-line between simple pulmonary distention and acute emphysema cannot always be positively determined.

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* *Virchow's Archiv*, vol. XVI; see also Ruge, *Berliner klin. Wochenschr.*, 1877.

TREATMENT.

The former hopeless views on the subject of the treatment of emphysema have long given way to the knowledge that a regeneration of distended pulmonary alveoli, even when the lesion is quite extensive, is possible, that elasticity can be regained; and that, even though old scars and atrophic areas cannot be removed, nature is ever striving by compensatory processes to repair the injury as completely as possible. It is therefore a most promising task to assist the reparative efforts of nature, or at least to remove anything that tends to disturb the processes of repair. The treatment of emphysema has developed along these lines until it now constitutes a complicated and difficult chapter in medicine.

A number of **prophylactic** measures are recommended by various authors. Individuals suffering from hereditary predisposition must use every means to strengthen the lungs from childhood on by proper diet and mode of life; they must avoid any occupation that necessitates the inhalation of dust and vapors, and must not become singers, preachers, orators, or performers on wind-instruments. Catarrh developing in such individuals must be treated seriously from the beginning. Affections accompanied by violent attacks of coughing require prompt and thorough treatment, and a sufficient period must be allowed for convalescence before the patient is permitted to return to work. An occasional visit to the south, the seashore, or the mountains should be urged for such patients early in their career. It is still impossible to protect our children against whooping-cough and other infectious diseases incident to childhood in the present questionable state of school hygiene; but even if the child does contract these diseases, much can be done to limit the evil consequences. To discuss this subject in detail would necessitate a complete review of the treatment of those diseases.

In addition to infectious diseases, chronic bronchitis and asthma demand especial attention. I must once more repeat my warning against the excessive use of drugs; they should be given only with the greatest caution and reserve. The ordinary expectorants, as a rule, have only a temporary value, and can be dispensed with altogether*; hydrotherapeutic procedures occupy the first place in the treatment. Hygienic measures cannot be too highly commended. The use of alcohol and tobacco must be prohibited absolutely. If it is possible to secure implicit obedience to these simple directions, the patient will himself combat the development of emphysema step by step, and is practically certain to escape a severe grade of the affection. That the disease nevertheless is destined always to be widespread is easily comprehensible for reasons that need no special exposition.

In the **treatment of pronounced emphysema** hygienic measures again occupy the first place. In a distinctly chronic disease like emphysema the mode of life is of immeasurably greater importance than any course of treatment that never lasts more than a few weeks, or at most a few months. It is possible in some cases to have the patient consider his infirmity in the choice of residence, but it is not to be supposed that any one locality possesses any special curative influence for emphysema. It was formerly the custom to advise high altitudes. It appears to me that a change of residence, the patient spending a good deal of time in various places, is to be commended as particularly useful. A cold, moist climate should always be avoided by emphysematous subjects; but after a sojourn in a mild and dry climate, a change to a mild moist region is often very beneficial, or, again, a change from a moist to a dry climate. A constantly mild and bland—that is to say, moist—atmosphere need only be recommended in cases of cachexia. The chief requisites are freedom from dust, therefore a sufficient distance from industrial centers, from important public roads, and all great centers of population. Hill-climbing is beneficial to a certain extent in emphysema; its value depends on the condition of the heart, which must be taken into consideration in advising its employment and specifying the amount.

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* This antiquated custom was condemned in the first edition of the "Allegemeine Therapie," but it will no doubt take some time to abolish it altogether.

The question of clothing may be considered in connection with the climatic treatment. On this point also there is a great diversity of opinion. Some recommend wrapping the body in wool; others prefer linen or muslin, and adapt their prescriptions to the season. I cannot subscribe to any extreme view. Individuals differ so much, and habit and mode of life necessarily influence the question so largely, that I cannot see the justice of prescribing definite rules in the matter of dress, as those who believe in exact prescription would have us do. It is quite proper to discuss the question of dress with the patient and to call his attention to manifest imprudences and mistakes, but one should not be tyrannical. Ordinary common sense is of more value in this connection than so-called scientific principles.

The patient must at least not be exposed to the danger of constant relapses by too much wrapping up and coddling. He must subject himself to a regular and systematic process of hardening, so as to acquire and preserve sufficient power of resistance against changes in temperature. If he is regular in the matter of sponging or bathing in cool water, spends a good deal of his time in the open air, and takes plenty of exercise, he is to a certain extent independent of dress, and is relieved of the constant worry of taking cold which in patients of this kind often becomes a perfect obsession; they cannot live without it and it robs life of all its pleasures.

The diet itself is also of considerable importance. It is not so much necessary to forbid certain dishes and recommend others, as it is to insist upon a proper moderation and to avoid any extravagance, or indulgence in unusual food. It would be quite wrong to assert that any one dish in itself is harmful. Every one should eat what agrees with him; in other words, he should regulate his diet so that he is free from abdominal discomfort and goes to stool regularly; only he must not eat more than is absolutely necessary. This rule is often and flagrantly violated, and so the physician is often forced to prepare a diet list with quantitative prescriptions. This applies especially to cases characterized by a tendency to obesity, which are quite frequent. The amount of fluid to be taken must also be regulated according to general therapeutic principles. In difficult cases a complete change in the entire mode of life, as carried out in many of the well-known sanatoria, offers the only hope of success. A strict milk diet sometimes gives brilliant results in patients who have been addicted to overfeeding, as is shown by an instructive and interesting case recently reported by Lenhartz.*

The disease has also been attacked directly by means of physiologic methods of treatment. The most important of these methods consists in the use of the *pneumatic cabinet.* Cabinets of this kind are now found in various cities, and several writers have given us the results of their observations in such places in carefully prepared monographs. The work of Vivenot forms the foundation for everything that has been done in this field. Other material additions to our knowledge are due to the vast amount of original work that has been done in the Jewish Hospital in Berlin under the guidance of Lazarus.† In a cabinet of this kind the air pressure is gradually raised during the first half hour. The patients then breathe air at a pressure of 1½ to 2 atmospheres, from one-half to one hour, and finally another half-hour is consumed in bringing the pressure back to the normal. The latter precaution is very necessary, as rapid increase of pressure and, still more, rapid decrease of pressure have often led to disastrous results.

A good many emphysematous patients experience great relief from the treatment, but the difficulty of explaining this good effect is still considerable. It has been known since the time of Vivenot that the depth of the respiratory movements is increased in the cabinet, while the frequency of respiration is diminished. These effects persist after the patient has left the cabinet, and, by repeating the sitting often enough, they may be made to continue for some time.‡

The effect on blood pressure is neither great nor constant. It is certain, at least, that the pressure is not diminished. The painstaking and laborious investigations of Liebig are absolutely depressing in their dearth of results. The effect on blood distribution is favorable, inasmuch as the blood is withdrawn from the peripheral portions, and tends to accumulate in the inner portions of the body. The effect of a séance on the absorption of oxygen and the elimination of carbon dioxid, as well as on metabolism, is so insignificant that it has not been possible so far to obtain any definite results in this matter. It was thought that the effect of the pneumatic cabinet might be enhanced by placing the patient in compressed air and having him expire into free atmospheric or rarefied air. Pircher has reported valuable results by this method.* The effect of expiring into rarefied air, while the patient himself is in compressed air, is said to be equivalent to negative pressure in the lungs, and even a small difference, of, say, 0.2 atmosphere, is sufficiently efficacious to obviate the necessity of risking the dangers incident to a great difference in pressure. I am not aware, however, that this method is carried out systematically anywhere at the present time. The investigations carried out at the Jewish Hospital and the work of Schreiber throw some light on the value of this method. They will be referred to in another place. But favorable results have been reported even without this feature of expiration into rarefied air; for instance, in Liebig's case,† which was one of fairly recent emphysema associated with asthma.

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* "Handbuch der praktischen Medicin," by Ebstein and Schwalbe, vol. I, p. 199. (A woman sixty-seven years of age, a teacher by profession, was affected with bronchitis and emphysema, edema in various portions of the body, albuminuria, and intense subjective symptoms. Temporary relief only was obtained with digitalis. After a diet consisting of 200 c.c. (say, 6 ounces) of milk four times a day, continued for six days, a permanent improvement took place. Dyspnea, edema, and albuminuria disappeared altogether.)

† Lazarus, "The First Twenty-five Years of the Pneumatic Institute."

‡ Aron denies this at present.

It appears, then, that the *indications* for the employment of these cabinets cannot be deduced or even controlled by scientific reasoning, but must depend on empiric results as reported by the experienced men in charge of institutions of this kind, among whom Lazarus, at the present time, is to be regarded as the chief authority. He is extremely guarded in his opinions, and absolutely refuses to admit patients with rigid thorax and arteriosclerosis into his apparatus, so that a large proportion of emphysematous patients is excluded from the method. The cabinet is indicated in the case of catarrhal conditions of the bronchial mucous membrane, and the good results obtained are inversely proportionate to the degree of pulmonary distention that has already developed. Increased pressure within the bronchi facilitates their expansion and acts as a stimulant to the torpid character of the chronic catarrh. Of course, the distention of the pulmonary tissue cannot be influenced. In a pronounced case of emphysema the compressed air treatment is of no value. It is more likely to be useful in cardiac forms accompanied by congestive hyperemia of the bronchial walls, the removal of which has a valuable reactive effect on the circulation of the heart and lungs. Such are the views of Lazarus. I have the impression that even arteriosclerotic emphysematous subjects often experience great relief in places like Reichenhall, and am therefore inclined to take exception to the dictum that in a pronounced case of emphysema the compressed air treatment is of no value.

For a time the hope was entertained that this method would furnish a remedy for emphysema, because it was thought possible that an increase in the absorption of oxygen might be brought about during the procedure; but, to judge from the great number of observations now at our disposal, this does not seem probable; although it possibly does take place to a very slight degree, the benefit is then correspondingly small. If it is desired to induce a greater absorption of oxygen in the body, direct *inhalations of oxygen* can now be resorted to. The gas can be obtained anywhere compressed in cylinders, and is readily inhaled by the patient with the aid of a suitable mask.

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* *Wiener med. Wochenschr.*, 1877, Hill.

† *Wiener med. Presse*, 1876.

Michaelis lately collected a number of data from Leyden's clinic* bearing on the use of this method, and his results are such as to encourage further experiments. When the general appearance and habit of the patient betray the want of oxygen, especially when pronounced cyanosis is present, these artificial inhalations may be resorted to. Michaelis observed a complete change to come over a cyanotic face with dark, bluish lips after the inhalation of 20 liters of oxygen; the lips became bright cherry-red and shining, and the face assumed a fresh, rosy look. The anxious look, which had been marked before on account of the dyspnea, gave place to a pleasant expression. In addition, the use of the method has a psychic effect. The patients believe in the remedy; in fact, they as a rule entertain extravagant hopes, and so it affords an excellent symptomatic measure. If the inhalations of oxygen are persisted in for some time, it is quite possible that a favorable effect is produced on the heart and on the nervous system, and in this good effect the lungs will also share to some extent. Whether the treatment has any permanent influence on the disease still remains to be seen.

To return to the physical treatment of emphysema, there remains to be considered the apparatus of Waldenburg and others like it, a method which is called by Lazarus the *active pneumatic method,* in contradistinction to the cabinet treatment, which he calls the passive pneumatic method.

Waldenburg is chiefly responsible for the development of this method and promised wonders from its action. The portable apparatus which usually bears his name, although it was used long before his time and has been improved by other men,† is generally familiar, and need not be described here. The patient inspires moderately compressed air, at an excess of $\frac{1}{40}$ to $\frac{1}{90}$ of an atmosphere, and expires into the normal air of the surroundings. Debilitated individuals should take a long rest after consuming the first cylinder; later, the dose may be increased to three or even five cylinders. When the catarrh begins to subside and the irritability of the bronchi is not great, the patient may expire into rarefied air ($\frac{1}{80}$ to $\frac{1}{60}$ of an atmosphere), using one to five cylinders interrupted by periods of rest. Great benefit was expected to result from this method. On theoretic grounds it is evident that the air is, as it were, drawn out of the thorax when the patient expires into rarefied air, and that the tension of the alveoli is thus diminished. The succeeding inspiratory effort must accordingly be facilitated, and the circulation in the capillaries of the lungs encounters less resistance. The portions of the lungs that are merely ectatic and have not yet undergone atrophy, are said to regain their elasticity by this means.

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* *Zeitschr. für diätetische und physikalische Therapie,* vol. IV, No. 2.

† Geigel-Mayer exhaust-wheel ventilator, for instance.

For these theoretic results, which appeared *à priori* plausible, Waldenburg later brought experimental support, but his beautiful structure collapsed so ignominiously that it is impossible to believe that his studies of the subject were thorough. His bulky volumes now stand neglected and covered with dust on the shelf, and no one dares to touch them. Very soon after their appearance, skeptics and critics made themselves heard. In a most commendable little monograph by Josephson, entitled "Wirkungslosigkeit und Nachtheile der transportablen pneumatischen Apparate von und nach Waldenburg," Hamburg, 1877, the coarser errors of that author are exposed, and other authors are cited who oppose his views; but the authority of the eminent clinician and of his followers sufficed to secure recognition for his work for some time.

The merit of clearing up the question in all its bearings is again due to the work performed under the inspiration of Lazarus at the Jewish Hospital at Berlin. It was found, by having healthy subjects expire into rarefied air,* that an increase in the rarefaction of the air was accompanied by a decrease in the vital capacity and of the quantity of carbon dioxid.† The same experiments were then repeated on emphysematous subjects,‡ with the result that neither the volume of expired air nor the elimination of carbon dioxid was increased; in fact, the results were the same in every respect. These findings are confirmed by the results of other investigators. J. Schreiber expended much time and effort on this subject.§ Although I am not prepared to admit the correctness of his first calculations in regard to the inspiration of compressed air, the experiments which here concern us particularly are those performed with the expiration into rarefied air, and the results of these harmonize in every respect with those obtained by Lazarus. Schreiber, however, finds that in the case of emphysematous subjects the quantity of air abstracted from the lungs during expiration into rarefied air exceeds by about 150 c.c. the quantity abstracted during expiration into the atmosphere. With the aid of a stethographic examination and certain other observations he then explains why even this small result is only apparent, and does not depend on an actual diminution in the capacity of the thorax, which is absolutely enlarged. The value of the procedure, he says, consists in the removal of acute and subacute alveolar ectasia only. It follows, then, that Schreiber considers expiration into rarefied air to possess a certain value if the acute or subacute alveolar ectasia of which he speaks is the condition which, as emphysema progresses, must eventually lead to the constantly increasing enlargement of the alveoli. Would not the method, therefore, be a valuable auxiliary measure to counteract the development of emphysema? On the other hand, Schreiber admits that there are certain drawbacks: (1) The decrease in intrabronchial pressure brought about by expiration into rarefied air must cause a more abundant afflux of blood, which appears to be practically indispensable in emphysema*; (2) the results of the treatment are often only evanescent, and disappear when expiration ceases. In severe cases expiration into rarefied air may do harm by increasing the work necessary for respiration.

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* Lebegoff.

† Schlesinger.

‡ Lanz. Randazzo, *Med. Centralblatt*, 1887, No. 12.

§ *Zeitschr. für klin. Med.*, vol. XIII.

The practical result of all these reservations would seem to be that an emphysematous subject should not be allowed to undergo the treatment. If it is desired to use it prophylactically in acute and subacute distention of the lung, there may be no objection under favorable circumstances and in the case of sensible persons in fairly good health who have time and money to spend. In most cases, however, the method is too expensive and too uncertain, and is not as effective as simpler methods. I am inclined to prefer the latter, particularly as I cannot help thinking that very unequal effects must be produced in different portions of the lungs by the employment of the apparatus. The portions that are weakest and most diseased are probably benefited least, or possibly even affected unfavorably, and I have the distinct impression that more harm than good is done with these artificial methods of treatment.

If, then, there is some virtue in the use of Waldenburg's apparatus, —and in view of the numerous reports of favorable results and the enthusiasm that prevailed at the time I am not prepared to deny it,— the success is to be attributed in part to suggestion and in part to the fact that the patients, in using the apparatus, go through a kind of *respiratory gymnastics* which we are still constantly in the habit of using with as much benefit as confidence. The beginning of respiratory gymnastics consisted in simple compression of the thorax. Gerhardt † proposed to have the patient go through respiratory exercises while an assistant, during expiration, compressed the inferior and lateral portions of the thorax. The proposition was well received, and has since been further developed. It later led to the construction of the breathing chair of Rossbach. The patient sits on this contrivance and by means of two levers to which broad straps are attached compresses the lower portion of the thorax at each expiration.‡

Ivanhoff§ used Rossbach's chair in the treatment of ten emphysematous patients, and reports that the circumference of the thorax was diminished on the average by 0.5, while the pulmonary capacity was increased by 350 to 930 c.c.

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* Some authorities consider the stimulation of the circulation an advantage, and personally I am not inclined to regard it with suspicion.

† *Berliner klin. Wochenschr.*, 1873.

‡ A full description of this apparatus will be found in *Klin. Centralblatt*, 1887, p. 31, Supplement.

§ Dissertation, St. Petersburg.

Strümpell recommends a simpler and much cheaper apparatus consisting of two boards joined at one extremity by an elastic band. The boards are applied to the sides of the thorax, the patient takes hold of the anterior extremities and brings them together at each expiration, thus compressing the thorax laterally. Langerhans * added a pneumatic rubber padding to the inner surface of the boards and used the apparatus extensively in my clinic.

Schreiber recommends uniform compression of the thorax by means of elastic corsets. The abnormal emphysematous breathing he claims is thus rendered practically normal. Expiration is facilitated and accelerated, inspiration is somewhat prolonged, and the respiratory pause make its appearance. He shows by means of mensuration and spirometry that by wearing the corset a diminution of the thorax is brought about which exceeds that which can be produced spontaneously in the expiratory position. This therapeutic effect can be temporarily enhanced by simultaneous expiration into rarefied air.† Another form of compression by means of a girdle was practised by Steinhoff.‡

As early as 1883 Feris § recommended a bandage similar in form to a double hernial truss; the two pads were so applied as to exert pressure in front on the upper intercostal spaces, and in this way are said to diminish the dyspnea. I have had no experience with these various forms of compression apparatus nor have I ever met any emphysematous patients who had worn them permanently or had anything to say in their favor.

The dangers that have been attributed to this method—namely, a tendency to hemoptysis, vertigo, and albuminuria—are to be dreaded only when the manipulations are performed carelessly or with excessive violence. Simple compression, as advised by Gerhardt, has long been abandoned, but respiratory exercises are destined to remain as a valuable therapeutic aid. None of these mechanotherapeutic methods are to be employed in very old or very feeble patients, or in persons with cardiac disease or a tendency to hemoptysis.

I am convinced that with the persistent use of respiratory gymnastics, supplemented by moderately active hydrotherapeutic measures, adapted to the age and strength of the patient, much good can be accomplished. These principles, which I laid down in the first edition of my lectures on "Allgemeine Therapie," will be found more fully elaborated and emphatically inculcated in my numerous later writings. Generally speaking, the use of any apparatus, and particularly the

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* *Zeitschr. für diätetischz und physikalische Therapie*, vol. II.

† I take exception to this statement for reasons already explained.

‡ His method is fully described in *Berliner klin. Wochenschr.*, 1890, No. 40, and additional observations are found in the same periodical, 1892, Nos. 31, 38.

§ *Société de thérapeutique*, July 12, 1883.

more complicated ones, is to be deprecated. People in easy circumstances may be advised with a clear conscience to invest in them, and they will no doubt use them with enthusiasm for a few days or even weeks, but before long, when the hoped-for miracle fails to be realized, the apparatus is relegated to the lumber-room. The essentials in respiratory gymnastics, which any educated physician can deduce for himself from our anatomic and physiologic knowledge, have been collected in an attractive form by Hughes,* and his book may be placed in the patient's hands for the purpose of educating him in an intelligent and persevering employment of the method. Landerer † also gives a full description of respiratory gymnastics. The simplest form consists in deep breathing. In the case of emphysema the accessory movements that are especially adapted to assist expiration are most important; all tight clothing is to be removed; the exercises should be performed only with the mouth closed. The fundamental rule is, that movements in which the arms are moved away from the chest assist inspiration, while movements in which the arms are brought near the chest assist expiration, whether the movements be active or passive. In ordinary cases the patient may hold a light dumbbell, weighing about one pound, in each hand and raise the arms from the ordinary position at the sides to the horizontal position; the hands are then brought into supination, palm upward, and during this movement an inspiration should be taken. The same movements are then repeated in the inverse order until the arms are brought to the sides of the body, and the patient then bends the knees until the dumbbells touch the floor. During this movement an expiration is performed.

In the beginning of the course the patient must rest even after a single movement, and I have come to regard six inspiratory movements of this kind as a very respectable performance that should not be exceeded by most emphysematous patients. In many cases it is better not to order the entire movement, but to use only the elevation of the arms or the bending of the knees. If the movement is confined to elevation of the arms, a staccato expiration consisting of two or three brief vigorous contractions of the abdominal muscles is to be recommended at the end of the ordinary expiration.

The number of movements to be performed is to be accurately prescribed by the physician. To insure their being carried out regularly and correctly, the patient must go through his exercises regularly under the physician's supervision. After a time, when the patient has been fairly well trained, the physician may give him a similar form of exercises to go through at different periods of the day when the physician cannot be present, the frequency of the movements and the duration of the pauses to be accurately prescribed. But he must always retain a sharp control, as later on the patient is inclined to sin quite as much through neglect as he was in the beginning to overdo the exercises. In all the larger cities institutes are now found in which exercises of this kind can be carried out successfully under a competent teacher. Among these institutions are the Zander institutes, and exercises with Zander's apparatus may often be added to the treatment with advantage if cardiac weakness is to be combated at the same time. The principles that underlie the exercises directed to the invigoration of the heart will not be discussed in this place, but must nevertheless always be borne in mind.

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* "Lehrbuch der Athmungsgymnastik," Wiesbaden, 1893.

† "Mechano-Therapie," Leipzig, 1894.

The following method,* recently proposed by Gerhardt, may be found useful in certain cases: The patient lies prone with the arms crossed on the back and the soles of the feet pressed against the foot of the bed, or with the toes buried in the mattress; a small pillow is interposed between the upper portion of the chest and the mattress, while another pillow supports the forehead. The patient is told to breathe deeply and at each expiration to extend the ankle-joint forcibly so as to press the chest against the pillow. This method reminds me of the history of an emphysematous patient of Schmidtlein's who always felt most comfortable when he was lying on his stomach. As a rule, these patients do not like the prone position, so that the method of Gerhardt will probably not be applicable in a very large circle of patients.

In the constant endeavor to improve on the results obtained with expiratory gymnastics, massage of the thorax was gradually added. It is undeniable that all kinds of unpleasant subjective symptoms can be removed by this procedure. When there are rending or lacerating pains in the chest, massage is often of great advantage; but the extravagant claims that the circulation in the rigid walls can be improved and their elasticity restored are quite properly received with suspicion. If the patient is wealthy enough to afford it, there is no objection to trying what a prolonged course of massage will do. As a rule, subjective symptoms are greatly improved, the patient himself is satisfied, and the suggestion of the masseur, who is naturally interested in the success of the treatment, is not to be despised either. Other authorities even maintain that a good effect is exerted directly on the circulation in the lungs, but I think this is a mistake. Such an effect might possibly be produced with the aid of concussion massage, which is performed with the so-called "concussor." With this apparatus, which is unquestionably not without value in physical therapeutics, and which I have often used with great advantage in the public clinic, it is possible to bring about a number of small concussions in rapid succession that are not confined to the surface but also affect the deeper tissues. Its use is not attended by any discomfort or pain, as is frequently the case in other forms of massage; on the contrary, the patients assert that the concussion is agreeable and makes them feel better. Its effect on the thorax is quite as favorable as that of the ordinary method of massage, and it undoubtedly exerts more influence on the pulmonary tissues, at least on the superficial layers, so that it is quite possible that it may stimulate the circulation and favorably influence the nutrition. Next to respiratory gymnastics, I should consider this method as specially worthy of thorough and persevering trial.

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* *Zeitschr. für diätetische und physikalische Therapie*, vol. I, No. 1.

A more trustworthy, not to say absolutely indispensable, auxiliary in the treatment of emphysematous patients is found in **hydrotherapy.** I have long been in the habit of sending these patients to hydrotherapeutic institutions and find the practice very satisfactory. They usually carry away with them one or more of the procedures that have been used on them, choosing the one that they find most agreeable, and use it again and again at home. Brief affusions and douches at various temperatures, and a cold rub or partial packs, are always useful and are often followed by brilliant results. The good effects achieved are readily understood. It is to be borne in mind how important for the emphysematous patient is the favorable influence on the heart which always attends hydrotherapeutic procedures.

Compresses and inunctions to the thorax are employed partly to alleviate pain and partly to combat dyspnea: For the alleviation of pain warm poultices, the Priessnitz compress,* and inunctions of chloroform oil and salicylic acid † are chiefly used, and are found to give the patient great relief. Dyspnea is treated with mustard plasters and occasionally with the aid of acupuncture.

Mineral waters play an important part in the treatment of emphysema. Their chief utility depends on the same principles that have induced me to recommend them in bronchitis, and the reader is referred to what I have said in that connection. In many cases direct laxative cures are recommended, and the patients are then sent to Carlsbad, Marienbad, Homburg, and other similar resorts. The patients for whom these waters are suitable are usually recognized at once by the fact that they have always been great admirers of aloes and rhubarb. Of course, these water cures have no specific influence on emphysema, but the large number of cases in which the emphysema is combined with obesity and the heart still retains its vigor are peculiarly adapted for this form of treatment.

Numerous medicinal agents are used in emphysema, but their effects are, of course, merely symptomatic; nevertheless they are often of great value, and in the advanced stages absolutely indispensable. What has been said in regard to drugs in connection with bronchitis and asthma should be carefully read, and as I should only have to repeat in the main what I have already said under that head, I shall cut the present description short and pass over the expectorants in silence.

Nux vomica is said to influence the smooth muscles, and this is undoubtedly true; but how long can we expect to lighten the burden of the bronchi by such artificial stimulation?

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* Or the Japanese warming-pan, the thermophore.

† Ten per cent. in vasogen (vasogenum spissum) to be rubbed in and covered with cotton. A. W. Muller, "Therapie der Gegenwart," 1899.

Digitalis is always a useful symptomatic remedy in advanced cases, in which the heart and circulation have begun to suffer. Strophanthus and caffein are also useful drugs in the same condition.

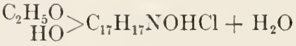
In the senile forms of emphysema alcohol is a valuable auxiliary remedy. Sometimes mild chalybeate preparations may be added with advantage.

Calomel, which was highly lauded by Stokes, finds its usefulness in the cyanotic form of the terminal stage. I regard it as a dangerous remedy, unsuitable in private practice. In the worst cases one always has to fall back on our old and tried narcotics, of which morphin is of course the most important. Numerous competitors have recently appeared; among them codein, heroin, peronin, and dionin.

Heroin has received as much praise as condemnation; after it had been found to act well in a series of cases, its use was occasionally followed by unpleasant and unlooked for after-effects, and, although even our best and most trustworthy remedies are not quite free from this reproach, it is especially necessary in the case of heroin to enjoin caution and to begin with small doses. It necessarily follows, however, that the effect must always be more or less doubtful.

Codein has been known for some time; it has recently been brought to our attention again by artificial means. It is always a valuable remedy in cases where there is reason to dread the effects of the continued use of morphin, and wherever a change is desirable. It can only be given internally, as subcutaneous injections are always very painful.

Dionin appears to be a remedy that deserves a good deal of interest; it is chlorid of ethyl—



$$\begin{matrix}C_2H_5O\\ HO\end{matrix}\!\!>C_{17}H_{17}NOHCl + H_2O$$

—easily soluble in water (1 : 7), alcohol, and syrup, and in doses of 0.002 to 0.004 ($\frac{1}{30}$ to $\frac{1}{15}$ grain) for children from three to twelve years of age and 0.002 to 0.02 ($\frac{1}{30}$ to $\frac{1}{3}$ grain) for adults, acts as promptly, on the average, as morphin; the dose must be increased gradually. In cases where morphin has already been used for some time, dionin loses its effect. Like morphin, dionin is very well adapted for subcutaneous injection. It is said to possess advantages over morphin in that it is not as likely to produce the drug habit; that a tendency to palpation and a feeling of pressure in the head are observed more rarely than in the case of morphin; and that it has a favorable effect on the night-sweats of phthisis (Jarisch), while morphin usually has the opposite effect. Jarisch prefers it to morphin in the treatment of emphysema and asthma, for the reason especially that, according to his experience, the amplitude of the respiratory movements is materially diminished during the use of morphin or heroin; while if codein is used, the depth of the respiratory movements is slightly increased, and during the administration of dionin the increase is still greater. Oxygen absorption and carbon dioxid production are increased in proportion to the increase in the respiratory labor. This result harmonizes with the interesting statements of Winternitz,* who asserts that heroin distinctly diminishes the irritability of the respiratory center, as shown by the fact that the amplitude and frequency of respiration are diminished, while codein or dionin do not produce the same effect. In this respect heroin acts precisely like morphin, in regard to which Filehue reports the same observation. One must not be misled by these theoretic results to say offhand that morphin is unsuitable in bronchitis, emphysema, painful cough, and conditions of dyspnea, and should be abandoned for dionin; such hasty conclusions are dangerous, as every one sufficiently knows. Niemeyer has rightly said that morphin is an excellent expectorant, and such it will remain in spite of all theoretic speculations. The superiority of dionin must be proved at the bedside, and cannot be deduced theoretically from laboratory experiments. I shall not say any more than that dionin and morphin are not quite equivalent, and that the former may in certain cases be the better drug. I believe, however, that morphin is certainly to be preferred in a conservative course of treatment, and to use dionin as a supportive measure in connection with mechanotherapy seems to me absolutely wrong. Jarisch's words, "especially in the mitral stages of phthisis and when codein or dionin often have to be used for considerable periods," suffice to show that we are antipodes so far as therapeusis is concerned. On the other hand, it would appear from the practical results obtained by Körte,† Freymuth, and Jenisch that dionin, so far as we know, should find a place among symptomatic remedies to be recommended in severe cases of emphysema for the purpose of further trial. The use of paraldehyd is distinctly to be deprecated.‡

In the cyanotic form of the terminal stage puncture of the lower extremities, if they are swollen and edematous, is often followed by great relief, and is therefore to be recommended in all cases even if it only has the effect of prolonging life by a short period. In very cachectic patients puncture may directly hasten the fatal termination.

The older physicians sometimes recommended venesection for the relief of marked congestive phenomena in patients that were not too greatly debilitated, and the procedure is perhaps not without some value.

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* "Therapeutische Monatshefte," 1899.

† "Therapeutische Monatshefte," 1899.

‡ Rolleston, *Med. Centralblatt*, 1889.

COMPENSATORY EMPHYSEMA.

PATHOGENESIS.

This condition cannot be regarded as an idiopathic and well-defined disease. A great variety of pulmonary diseases in which portions of the bronchial tree are almost or quite impermeable to air are attended with the production of circumscribed emphysema, just as diffuse disease of the bronchi is followed by the production of diffuse emphysema. But as it appears obvious that distention of the lung takes place in portions that were not primarily diseased, and that the purpose of this distention is to fill out the vacuum which is left by the failure of the diseased portion to expand, the conception that the distended portions perform the work that the diseased portions fail to do suggests its nature, and thus the name compensatory emphysema was introduced. The essential part of the definition, therefore, is that tissue which was previously healthy has become emphysematous. The direct result of this definition is the view that the elasticity of the lung can be permanently injured simply by excessive inspiratory efforts, a view which I was unwilling to accept in the case of primary emphysema.

In this connection, also, I adhere to the stand I have taken. Even though this view corresponds in the main with Andral's exposition of the matter,* and has found many supporters, I do not regard it as at all certain that the distention really affects portions of the lung that are absolutely sound. Such a view would imply (1) that circumscribed diseases may exist in the lung without influencing the integrity of neighboring parts, a possibility that seems to me extremely improbable; (2) that the organism regulates the degree of respiratory effort according to its need for oxygen without any regard to the condition of the lungs, an assumption that also offends my notions of biologic processes.

That the expanding thorax is not entirely independent of the distending power of the lungs, and that the mechanism must be provided with some regulating apparatus, is evident, for example, in many cases in which one side or part of one side remains immovable during respiration because the portion of the subjacent lung is infiltrated and cannot expand. This occurs, for example, in cases of recent pneumonia where the presence of contraction or adhesions is still out of the question.

I do not, therefore, admit that the unqualified inspiration theory is sufficient to explain the production even of compensatory emphysema; however, I am willing to admit that it is relatively most important in this form of the disease. I will also admit that cases of sudden and profound disturbances may sometimes occur in which the utmost efforts at compensation and correction are inadequate, and the course rapidly follows the schedule of compensatory distention. This happens, for example, when a large portion of the lung suddenly loses its power of distention, so that the entire negative pressure (suction power) of the thorax is exerted on a comparatively small portion of the lung, as in sudden occlusion of the main bronchus, embolism in a branch of the pulmonary artery supplying a large portion of the lung, and extensive infiltrations of very sudden development. But even such cases are not free from suspicion, as it is not probable that the portions of the lung that are not involved in the disease escape without edema or some marked circulatory disturbance; and it is well known that any form of edema leads to acute pulmonary distention.

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* Andral, "Clinique med.," vol. III, 1st edition, p. 62; see also Gairdner, *Archives Generales*, 1854, vol. II.

I myself saw a case of mediastinal tumor in which the growth of the neoplasm rapidly produced occlusion of the main bronchus; the other lung became distended so rapidly as to suggest the presence of pneumothorax.

The rule is that compensatory, like primary emphysema, is produced by various injuries, often acting together, during both inspiration and expiration, and by disturbances of the circulation. It is therefore not characteristic of this particular form of emphysema to develop in sound tissue; only, as a rule, a focal disease exists in the lung which dominates the clinical picture and determines its nature. The reaction of this focal disease on the remaining, healthy tissue of the lung in a number of cases leads to the development of compensatory emphysema in the sound tissue through the medium of various conditions, such as catarrh or edema, that interfere with the free ventilation in the bronchi. Quite often it amounts to no more than an acute distention of the lungs developing in the last hours or, at most, in the last days preceding death. Such a condition is observed particularly in acute pulmonary edema, or in hemoptysis when the blood coming from the seat of the hemorrhage is aspirated into the various portions of the bronchial tree. Care is therefore necessary in interpreting an emphysema found at the autopsy.

The fallacy of the belief that this purely mechanical explanation applies in all cases is shown by the fact that in many individuals a large portion of the lung becomes atrophic in earliest youth without the remaining portions becoming emphysematous, as would necessarily follow if the theory were correct. Numerous cases of this kind have been described,* and the reports of Recklinghausen and Coats are particularly convincing. The sound lung becomes hypertrophic, and is thus able to equalize defects of considerable magnitude. The two sides of the thorax will appear equally well developed although one lung is wanting. Youth is probably an important factor in this mechanism. In advanced age a curative hypertrophy of this kind can hardly be expected; possibly such a condition may take place during the third decade of life. There is a complete dearth of exact observations on this point, although the question possesses considerable interest in connection with the cure of phthisis. Some therapeutists speak of hypertrophy, others of compensatory emphysema. The question is whether the former has really been clearly established; the latter is demonstrated daily at the autopsy table.

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* For an analysis of cases see Schuchard, *Virchow's Archiv*, vol. CL, p. 71.

In any well-marked case of tuberculosis of both upper lobes an increase in the volume of the lower lobes is usually present. The lungs invade the complementary spaces and force the diaphragm directly downward—for in reality they do force the diaphragm downward, although of course expansion is, in the first place, made possible only by an energetic inspiratory movement on the part of the diaphragm drawing the air outward into the lower portions of the lungs, and the depression of the diaphragm is due to the fact that the lungs fail to regain their original volume. This mechanism clearly shows that the enlargement of the lungs does not depend merely on the walls of the cavity,—the thorax and the diaphragm,—but represents an active movement, inasmuch as they are capable of resisting contraction. If they are able to do this, however, it is because of some lesion impeding the exit of air from the bronchi and alveoli, either an accumulation of mucus or a diminution in the elasticity of the tissues. This practically brings us back to the point of view which was defined in the discussion on diffuse emphysema; there is no sharp dividing-line between primary and compensatory emphysema, the two terms are mischievous in so far as they tend to maintain a one-sided view of the question.

VARIETIES AND DISTRIBUTION.

As emphysema manifests itself in various forms, a classification has been introduced.

1. **Bullous Marginal Emphysema.**—This occurs practically only at the edges of the lung, and may be characterized by the formation of blebs varying in size from that of a hazelnut to that of a lemon. They are frequently pedunculated and the walls are covered with a thick layer of pleura, but they are almost never adherent to the costal pleura.

2. **Lobar Emphysema.**—This develops in connection with atelectasis of an entire pulmonary lobe; does not, as a rule, attain a high degree of severity; and, more than any other variety, suggests that healthy portions of the lung have undergone distention because a large portion of the tissue has become contracted and lost its functional activity from disease. Cases of lobar emphysema are noted particularly in the atelectasis of kyphotic individuals, in chronic tuberculosis, in contractions and adhesions following extensive pleurisy, and in fibrinous bronchitis. The group also includes the

emphysema which in children suffering from pneumonic disease of the posterior and inferior portions of the lung develops in the anterior and upper portions of both lungs, especially when the dyspnea is great. It is also caused by other lung affections in children. Numerous examples are cited by Hervieux,* and Rilliet and Barthez have also observed it.

Rachitic changes in the walls of the thorax, like kyphosis, are almost regularly accompanied by emphysema of large portions of the lungs which in their development rationally follow the compensatory dilatation of the thorax. The term compensatory emphysema is applied to these forms with special preference.

3. **Lobular Emphysema.**—This occurs in the tissue surrounding small multiple foci, and possesses only a pathologic interest.

It is met most commonly in tuberculosis; it also occurs in chronic bronchopneumonia; in peribronchitis, particularly in the variety due to pneumonokoniosis; in chronic pleurisy and its remains; in tumors; in atelectatic foci of limited extent, such as particularly accompany valvular disease with brown induration. It is seen most rarely in connection with gangrenous foci or bronchiectatic cavities.

An exhaustive paper on the subject of emphysema in tuberculosis has been contributed by Gallard.† Compensatory emphysema manifests itself in a variety of forms in tuberculosis. A certain interest is attached to the condition from the fact that it has been thought by some to indicate a curative effort on the part of nature. I agree with Gallard that the effort, if it exists, is certainly not a favorable one. If a certain amount of lung-tissue has been lost and a vacuum is to be filled, depression of the walls of the thorax and elevation of the diaphragm will accomplish it much more successfully.

The acute pulmonary distention that sometimes develops in patients who die with the symptoms of intense dyspnea in the last days of life or during the agonal period must be distinguished as a special form. The usual cause is pulmonary edema. The rule already laid down in connection with primary emphysema may be followed here as well. An emphysema should be declared to exist only when the condition in question has lasted at least a week.

SYMPTOMS.

A slight partial emphysema in itself produces no symptoms. If it becomes extensive, the symptoms of diffuse emphysema make their appearance, the severity being in direct proportion to the extent of the disease. If it is confined to one-half of the lungs, as happens in some interesting cases, the symptoms must, of course, be confined to that region. I need not go into the details of the symptomatology again.

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* *Archives générales*, June, 1861. The paper is marred by the fact that vesicular and interstitial emphysema are grouped together.

† *Archives générales*, 1854, vol. II.

It is said that a circumscribed tympanitic area is always found when there is a small focus of emphysema; but I do not believe that this sign is often present. The word tympanitic is often used very loosely, and I am convinced that it is not infrequently incorrectly used.

PROGNOSIS.

The prognosis of partial emphysema is, of course, the same as for the diffuse variety, provided the emphysema is sufficiently extensive. A small area of emphysema is quite without danger. It may be said that whenever emphysema is extensive enough to be readily and positively demonstrated, it is harmful; if, on the other hand, it is so limited that its presence can only be inferred, it may be disregarded. As a general rule, these forms of emphysema are not followed by any sequels such as are observed after diffuse emphysema. Cardiac affections especially are not apt to develop.

The treatment is determined entirely by the dominant disease; the emphysema itself is practically neglected.

INTERSTITIAL EMPHYSEMA OF THE LUNGS.

PATHOGENESIS AND ETIOLOGY.

INTERSTITIAL or interlobular emphysema of the lungs is produced by laceration of the mucous membrane of a bronchus or laceration of the alveoli themselves. The air penetrates through the laceration into the connective tissue of the lung. If the surface of the lung is torn, a pneumothorax as a rule develops; but it is possible for the pleural investment to afford sufficient resistance to allow the air to accumulate underneath the uninjured pleura.* If the layers of the pleura are adherent, the air always remains underneath the pulmonary pleura. It is conceivable that it might also force itself into the tissues of the adhesions—that is to say, between the two layers of the pleura; but, so far as I know, there is no proof of this occurrence. Nor does the air collect underneath the costal pleura. If the laceration is near the root of the lung, the air may enter the mediastinum directly without going into the lung or pleura. This is the variety of emphysema that usually occurs in parturient women. It begins in a laceration at a definite point on the hilus, extends to the mediastinum, and ultimately reaches the neck. The cases reported by Quincke, Petersen, and Schotten † belong to the same variety. Mediastinal emphysema and general cutaneous emphysema, which is produced when in the presence of adhesions the lung ulcerates and a communication with the subcutaneous connective tissue is established, so that emphysema of the cutaneous integument is formed directly from the lung, will not be considered in this article.

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* Z. B. Grancher, *Union méd.*, 1886, No. 41.

† *Berliner klin. Wochenschr.*, 1884–86.

First and above all, the student must be warned not to mistake so-called cadaver-emphysema for one that has been produced during life. The pathologists have repeatedly called attention to the danger of this mistake.

Theoretically distinctions are made according as a laceration in the lung is produced by a traumatism, a morbid process, or excessive exertion during coughing. As a rule, the cough is the final cause, the pulmonary tissue having already become altered by traumatism or a morbid process. Nor can it be doubted that even in a perfectly sound lung violent cough is capable of producing emphysema. Owing to the great variety of cases that occur, some classification is indispensable, although cases will always be found that defy all attempts at subdivision.

Interlobular emphysema may be studied under the following heads.

1. *Emphysema due to traumatism in the narrower sense.*

(*a*) Traumatism acting from without, such as a stab wound, a blow, or contusion of the chest. The external cutaneous integument may be quite uninjured; the accident has been especially attributed to the effect of splinters from a broken rib, but there have been plenty of cases without injury to the ribs in which the force of the contusion alone sufficed to bring about the laceration.*

(*b*) Traumatism acting from within. A knitting-needle had entered the esophagus and had become wedged crosswise. After a short time signs of cutaneous emphysema developed. Two weeks later fever set in, the sputum became offensive, pneumothorax developed, and the patient died. At the autopsy there was found interlobular emphysema of the right lung in which the point of the needle was embedded.

2. *Emphysema due to forcible insufflation.* This case might be included in group 1, (*b*). Insufflation was formerly a common practice for the purpose of resuscitating infants born in a state of asphyxia. At the present time it would be difficult to find an obstetrician to indorse the method, but in adults the direct insufflation of air is still resorted to in desperate cases. It should certainly be employed with great care, and should not be the only means resorted to, but should be combined with the external methods of artificial respiration.

3. *Emphysema due to foreign bodies in the air-passages.*

4. *Emphysema due to pathologic products in the air-passages.* This

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* Holmes, *Lancet*, 1886, vol. I, p. 879. Hemoptysis, extreme dyspnea, complete absence of respiration, and immobility on the right side without any change in the percutory note! On the following day amphoric breathing (?); no sign of air or fluid in the pleura. Recovery with areas of thickening in the lung. Thaal, *Wiener klin. Wochenschr.*, 1898.

class includes principally tumors in the trachea and the false membranes of diphtheria.

5. *Emphysema due to external violence acting on the air-passages.* Strangulation may here be mentioned. It frequently leads to emphysema.

6. *Emphysema due to voluntary (morbid) spasmodic closure of the glottis.* This group includes the emphysema of parturient women, the emphysema in spasmodic closure of the glottis, and the emphysema following violent efforts of coughing. But as the paroxysm of cough is usually dependent on some definite injury, the cases ought really to be included under 1, 3, and 4; thus, for example, a paroxysm of cough following the inhalation of irritating vapors should be included under 3.

7. *Emphysema due to excessive inspiratory efforts.*

8. *Emphysema due to pathologic conditions leading to perforation of the lung,* such as ulceration occurring in phthisis, abscess, gangrene, and in connection with tumors and caseous glands.* This subdivision cannot always be separated from group 4.

Group 7 calls for special criticism. A case observed by Virchow may be cited in this connection. The man had shot himself in the head and, owing to pressure on the medulla oblongata, violent inspiratory movements had been produced. The doubts that I have expressed in regard to the possibility of inspiration alone having such an effect in connection with vesicular and compensatory emphysema do not apply here. There are places in the lungs that unquestionably are subjected to extreme traction in any forcible respiratory effort. They are found particularly where the pleuræ pass from the lung itself to the large bronchi and adjoining tissues. These areas are to be designated as *loca minoris resistentiæ.* Such areas may be produced by external causes, and it follows therefore that inspiratory overexertion alone must be recognized as a cause of interstitial emphysema, although I denied this causation in the case of vesicular emphysema.

As regards the pulmonary affections, it may be stated *à priori* that any affection of the lungs may occasionally lead to interstitial emphysema; but those diseases are to be particularly feared that give rise to violent paroxysms of cough. Interstitial emphysema is principally observed after phthisis, not, however, because the latter predisposes to emphysema, but simply on account of its frequency. Whooping-cough and bronchitis with intense paroxysms of cough, as well as dry bronchitis (*bronchitis sicca*), should also be enumerated among the more frequent etiologic factors. Bronchopneumonia, vesicular emphysema, or croupous pneumonia are generally regarded as more unusual causes, and next in order come the diseases which are rare in themselves. This point has already been discussed elsewhere.

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* See Marchand, *Prager Vierteljahrsschr.*, 1876, who gives examples of all these conditions.

That even a healthy lung is not absolutely secure from interstitial emphysema is shown by the cases reported in parturient women, in spasmodic closure of the glottis, and after the inhalation of irritating vapors. Ozanam * had three cases of subpleural emphysema of the unaffected lung in children with pneumonia.

In Thalma's † case a man aged thirty-five years was taken ill with a feeling of oppression in the chest, and fever. The latter gradually subsided; but dull areas remained in the lung, and in the fifth week emphysema made its appearance in the neck and below the clavicle. At the autopsy bullæ filled with gas were found underneath the pleura, in the pulmonary tissue and in the mediastinum. Tubercle bacilli were found in the consolidated portions. This was evidently a case of tubercular pneumonia with secondary interstitial emphysema—a condition that is unquestionably very rare.

Another case ‡ was that of a man with a severe vesicular emphysema who is said to have developed interlobular emphysema after an ordinary fall on the back.

In Guillot's case a phthisical individual, forty-two years of age, was suddenly attacked while at his work with violent dyspnea and intense pain in the left half of the chest. Four days later he became aware of a small soft tumor in the left infraclavicular region. The tumor rapidly grew, and extended to the face, the upper extremities, and the trunk. Crepitation could be elicited on palpation. The man died soon afterward and the left pleural cavity was found free from air, while the subpleural cellular tissue was emphysematous. At the root of the left lung there was a small opening whence the emphysema had extended into the mediastinum and continued to spread along the sheaths of the carotid arteries.

The mechanism is usually as follows: A small opening is formed in an alveolus, from which the air escapes into the tissue underneath the pleura or into the tissue surrounding one of the vessels coursing through it—at first only a small bubble the size of a hempseed. Multiple small openings are also said to occur. Under the influence of cough and forcible respiratory movements the beads of air multiply to form bright glistening lines like strings of beads which can readily be seen through the pleura in the interlobular tissue. The lobules are thus separated from one another by pneumatic spaces which are said to be wedge-shaped; Laennec compared them to the sections of an orange. The base of the wedge is directed toward the pleura. Gradually the pneumatic spaces may coalesce and form a large cavity. The pleura is separated from the underlying tissue over a large area and bullæ of considerable magnitude are produced. These bullæ continue to advance along the pulmonary vessels, which they dissect out in the same way as a dissecting inflammation. Thus the air reaches the large bronchi and enters the mediastinum along their walls. The pathologic picture may vary greatly, according as the air penetrates

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* *Archives générales*, 1854, vol. I, p. 47.

† *Zeitschr. für klin. Medicin*, vol. x.

‡ Saussol, *Gaz. des Hôpitaux*, 1882, p. 141.

into the deeper tissue or remains near the surface, the distribution depending on the position of the starting-point.

SYMPTOMS.

There are no well-marked symptoms in the lungs. Laennec was mistaken in saying that the presence of emphysema could be inferred from a resonant percussion note, or even that the bubbles of air under the pleura are capable of producing a dry friction râle. If the emphysema invades the mediastinum, it probably gives rise to a certain degree of dyspnea, but the phenomena are rarely as marked as they have been described in text-books, in which it is usually stated that a dangerous effect is produced by pressure on the heart and on the large blood-vessels.

COMPLICATIONS.

1. *Emphysema of the costal pleura.* This condition cannot be diagnosed, and little attention has been paid to it at autopsies. It would appear that the intimate attachment of the pleura as a rule prevents its production.

2. *Mediastinal emphysema.* As a rule, interstitial emphysema is not recognized until it has produced a considerable degree of mediastinal emphysema; the latter can be diagnosed, and from it the presence of interstitial pulmonary emphysema may be inferred. It must not be overlooked, however, that mediastinal emphysema may occur without pulmonary emphysema, even omitting cases of external injuries; thus, mediastinal emphysema may be produced by disease of other organs, the trachea, the right or left bronchus, the esophagus, or even the stomach.* The following symptoms are in favor of mediastinal emphysema:

A fine crepitant râle, usually synchronous with the cardiac action. Faber heard a crackling râle accompanying the heart sounds.

Disappearance or indistinctness of the cardiac dulness. Over the heart a sonorous, not distinctly tympanitic percutory note develops (F. Müller). Disappearance of the apex-beat.

The intercostal grooves near the sternum are obliterated or may even bulge.

3. *Cutaneous emphysema.* Emphysema is most readily recognized underneath the skin, where it gives rise to the characteristic crackling, elicited by palpation. It appears from the cases reported that cutaneous may be the result of mediastinal emphysema, and it may also be directly derived from the lung, the air having entered the thoracic wall through adhesions. The most frequent form of emphysema is that with "triple seat," as the Frenchmen call it—lung, mediastinum,

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* Case reported by Faber, *Würtemberger Correspondenzblatt,* 1885: "Perforation of a Gastric Ulcer."

and outer skin. Gallard collected numerous cases of this kind.* As most of the cases recover, the condition of the lung is not known, but it is assumed as a matter of course that the lung is involved when air reaches the skin from the mediastinum. But often enough the lung is quite unaffected. This is because the opening is very frequently situated at the root of the lung, where there is a weak spot, particularly favorable to the production of emphysema; it is the point of transition from the pulmonary to the costal, or, better, mediastinal pleura, on the anterior surface of the bronchus. At this point an alveolus is apt to rupture, and the air then passes directly into the mediastinum without diffusing itself in or upon the lung to any marked degree. It is, of course, obvious that cutaneous emphysema may be produced directly from the lung without the intervention of mediastinal emphysema, if the lung is adherent and an injury is produced at the point of adhesion. Thus, in one case simple puncture led to the production of cutaneous emphysema,† while in another case the air came from a cavity which was adherent to the chest-wall.‡

4. *Pneumothorax* is not very rare in extensive interlobular emphysema. The pleura gives way at some point, and air enters the pleural sac.§ It has also happened that emphysema, beginning in the hilus of the lung on one side and extending into the mediastinum and external skin, was in the end followed by the development of pneumothorax.||

5. *Entrance of air into the blood-vessels.* This is a much mooted point. Many regard the entrance of air, which is often discovered at the autopsy under these conditions, as a postmortem production, the presence of the air being explained either by fermentation or by aspiration through a vein at the time of opening the cadaver. There is no question that many accidents of this kind have misled investigators in the past; nevertheless there are cases in which it has been asserted to be highly probable that air from an emphysematous lung entered the vessels during life. There is no doubt that the condition has repeatedly been induced experimentally in animals by means of insufflation.** The question is especially important to medicolegal experts.

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* *Archives générales,* 1880, vol. II, See also vol. XIII of Nothnagel's "Encyclopedia of Practical Medicine"—"Diseases of the Mediastinum."

† *Lancet,* 1889, vol. I, p. 273.

‡ *Lancet,* 1889, vol. II, p. 1277.

§ Case of bronchopneumonia after measles, eventuating in double pneumothorax. Teissier, *Bulletin de la société anatome de Paris,* 1897, No. 17.

|| Grancher, *Union méd.,* 1886, No. 41.

** See Marchand, *Prager Vierteljahrsschr.,* 1876, vol. CXXXII, p. 119.

PROGNOSIS.

Interlobular emphysema is not, as a rule, dangerous, unless the individual is very cachectic, and weakened by the primary disease. I have seen a pneumothorax, which had evidently been produced in a case of interstitial emphysema by the laceration of subpleural bullæ, end in recovery. Koranyi even reports two cases of this kind that ended in recovery. Death is of course possible from shock or from the pleurisy that follows; in very rare cases it may be due to the entrance of air into the blood-vessels. Even mediastinal and subcutaneous emphysema are less dangerous, on the whole, than might be supposed after reading the text-books. In the very worst cases it may be justifiable to make small incisions to relieve the tension of the skin; but too much must not be hoped from the procedure except to alleviate the patient's sufferings.*

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* Torday, *Klin. Centralblatt*, 1886, p. 270. Rapid cure of an extensive subcutaneous emphysema after diphtheria. Maschner, *ibidem*, p. 125.

ATELECTASIS.

The doctrine of atelectasis was a serious stumbling-block to the older authors. The condition was often regarded as inflammatory, and was confounded with pneumonia. It was not until the year 1832 that the condition was properly interpreted in a dissertation by Jörg. A historical abstract and the bibliography of the old literature will be found in Hertz's "Handbuch."*

The term atelectasis (ἀτελής, "incomplete"; ἔκτασις, "dilatation") is applied to a condition of the pulmonary alveoli in which the latter are collapsed and almost or quite airless. No air enters during inspiration and the alveoli take no part in the function of the lung.

This condition is normal during fetal life; but if it persists to any degree after birth, it constitutes a pathologic condition. Such cases form the group known as *congenital atelectasis*, or *fetal condition* of the lungs. It is distinguished from *acquired atelectasis*, which includes the cases in which from one cause or another air ceases to enter the alveoli, while the air contained in them undergoes absorption and the alveoli thus become airless.

Acquired atelectasis is subdivided into atelectasis due to obstruction of the bronchi, and that produced by external pressure on the lungs, the two varieties being termed respectively *obturation*, and *compression* atelectasis. The term "pulmonary collapse" is often employed, but it is not quite synonymous with atelectasis, as it is used to designate a temporary condition developing very rapidly and not accompanied by any changes in the lung tissue.

Feustell suggests the following classification: (1) persistence of fetal condition of the lungs—*atelectasis;* (2) return to the fetal condition under the influence of forces inherent in the tissue—*collapse;* (3) return to the fetal condition under the influence of forces acting on the tissues from without—*compression.* This classification does not harmonize with the generally accepted meanings of the terms used, and is of no particular value, as the boundary-line between 1 and 2 is very indistinct in practice.

The subdivision of atelectasis into the congenital and acquired forms, which theoretically appears so natural, presents many difficulties, because in many cases the infants at first breathe quite satisfactorily and only after a time become apneic. A bronchus becomes occluded with mucus or particles of meconium, and a lung, which at first expanded normally, collapses and on the next day is found to be the seat of an extensive atelectasis. Even later, during the first or even second year of life, atelectasis may be produced in debilitated individuals by the action of some severe disease, particularly one associated with fever and bronchitis. This form is practically the same as the atelectasis that is acquired immediately after birth. Nature, with her manifold clinical pictures, mocks all our efforts at classification.

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* Edited by Ziemssen, vol. v, p. 418.

I shall, however, distinguish the disease of the new-born from that of well-developed individuals, or, as we usually say, adults.

MORBID ANATOMY.

The atelectatic area may vary greatly in extent, ranging from a small isolated focus to an entire lobe; the entire lung may even be atelectatic. These areas present signs of shrinking; the smaller ones are depressed below the surface of the healthy lung; the color of the pleura is dark bluish, distinctly darker than the surrounding tissue. The section is brownish-red, and the cut surfaces retract, are dry, not granulated, and no air can be expressed from them. Atelectatic lung tissue sinks in water.

For some time an atelectatic portion of lung retains the power of regaining its normal volume after being artificially distended with air, but gradually profound changes take place in its structure. The walls of the alveoli become completely adherent, the connective tissue is increased in quantity, and the lung tissue is converted into an indurated mass that retains only the merest trace of lung structure.

The subsequent changes that take place in atelectatic lung tissue, when the condition has existed for some time, are still the subject of dispute among pathologists. That the fetal condition may continue for an indefinite length of time is proved by many findings, as, for example, those of Franke.* As a rule, however, sequels in the form of multiplication of connective tissue and bronchiectasis develop.† Schuchardt's ‡ case I regard as a typical case of this kind. But even more extensive changes are described, such as cyst formation (Grawitz §), and proliferation of cartilage (Heller ‖), but in such cases it is impossible to believe that one has to deal with a simple atelectasis. There must have been some developmental anomalies. But as developmental anomalies are often enough complicated by atelectasis, it will be readily understood how difficult it is to draw the line between conditions that must be regarded as a consequence of one or the other of these abnormalities.** The question of the presence or

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* *Deutsches Archiv für klin. Medicin*, vol. LVI.

† This is shown by Köstlin's case, *Archiv für physiologische Heilkunde*, VIII, 1849; and by Rokitansky and Cohnheim, "Allgemeine Pathol.," vol. II, p. 180.

‡ *Virchow's Archiv*, vol. CI.

§ *Virchow's Archiv*, vol. LXXXII, 1882.

‖ *Deutsches Archiv für klin. Medicin*, vol. XXXVI.

** Consult, for example, the collection of cases by Wollmann. A case of agenesis of the left lung with bronchiectasia, Freiberger Dissertation, Dresden, 1891. A absence of pigment is of considerable importance in the consideration of this subject. But this obscure problem is far from being solved. It may be stated that whenever much pigment is present, the condition has not developed before the fifth to the seventh year. On the other hand, however, it cannot be asserted that in cases in which there is no pigment, the condition is necessarily fetal; because there is no doubt that under certain circumstances the pigment may be absorbed in the course of time.

From a practical standpoint it is quite correct to distinguish cases of true atelectasis from cases with cyst formation; cases with proliferations in the bronchial tissues, especially the cartilage; and cases associated with profound developmental anomalies that cannot be satisfactorily explained.*

PATHOGENESIS.

In a certain proportion of cases no air enters the alveoli, either because the access is completely cut off, as in many malformations, or because the obstacles which are present are too great for the infant's strength to overcome; the lung therefore retains its whitish fetal condition. In another series of cases the lung becomes atelectatic from compression; the air is simply forced out and cannot reenter. Tumors and exudates that compress the lung regularly lead to the production of circumscribed areas of atelectasis in the adjacent portions of the lung. Hypertrophy of the heart, deformity of the vertebral column and of the thorax, distention of the abdomen with forcing up of the diaphragm, and even simple meteorism, are to be included among the causes of atelectasis. In debilitated patients the mere dorsal position, if continued too long, suffices, by allowing the pressure of the abdominal organs to act on the posterior lower segments of the lung, to produce atelectasis, the strength of the respiratory movements being inadequate to bring about expansion. On account of the greater weight of the liver, the atelectasis produced in this way is much more pronounced on the right than on the left side.

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similar case is that reported in the *Lancet*, 1885, vol. I, p. 1048: The right lung appeared to be completely wanting in a man sixty-three years of age After a long search a rest, the size of a sausage, was discovered. The heart was displaced far to the right by enormous enlargement of the left lung. Malqueen's case in the *Lancet*, 1899, vol. I, p. 111, presented the anomaly of a left bronchus obliterated at the bifurcation, with enormous enlargement of the left lung; in the apex was a phthisical cavity from which the hemorrhage that caused the patient's death in his forty-third year was derived. Sieveking, in *Münchener med. Wochenschr.*, 1895, No. 4, reports the case of a man fifty-six years of age in whom the left lung was hard, airless, and of the size of a child's fist. The lower lobe was yellowish-red and free from pigment—fetal atelectasis; the upper lobe presented a slaty pigmentation.

* See vol. XIII, Nothnagel's "Specielle Pathologie und Therapie," and Fürst, "Missbildungen der Lunge," in Gerhardt's "Handbuch."

Rosenbach * has pointed out that in so-called compression atelectasis it is at first not a true compression that brings about the condition, but rather a force which, counteracting the expanding pressure within the thoracic cavity, enables the elasticity of the lungs to contract those organs to their smallest volume. It is only when complete collapse of the lungs has been brought about, and the pressure in the pleural sac is still further increased, that the lung actually becomes compressed, and the compression acts less on the parenchyma itself, which has already become contracted to its smallest volume, than on the vessels and on the bronchi.

The third group is formed by the cases of so-called obstruction atelectasis. When a bronchus is occluded so that no air can pass, the air in the spaces which it supplies logically undergoes absorption; first the oxygen, then the carbon dioxid, and lastly the nitrogen.† Lichtheim demonstrated that when a bronchus really becomes occluded, the portion of the lung which it supplied, as would be expected, becomes completely airless within a few hours. These experiments suggested to him the important assumption that the elasticity of the pulmonary parenchyma continues in its efforts to distend the organ until the last alveolus has yielded; and showed that the absorption of air is effected by the circulating blood. He calls attention to the fact that this absorption atelectasis is responsible for those cases of pleurisy in which the lung is not excessively compressed by a massive pleural exudate. The capillaries become dilated and the atelectatic portions hyperemic. These consolidations occur mostly in the deeper portions of the inferior lobe, and extend upward along the vertebral column in the form of a band about 5 cm. in width which gradually becomes narrower toward the apex of the lung. In time, however, serum is exuded into the alveoli, they become edematous, and the atelectatic area may actually bulge and show the signs of inflammation. This is the form of splenization or hepatization in which the boundary-line between edematous hyperemic atelectasis and bronchopneumonic infiltration is difficult to establish.

The atelectasis accompanying pneumothorax has excited peculiar interest. In the cases characterized by distinct excess of pressure in the pleural cavity (valvular pneumothorax) there is evidently a compression. On the other hand, in the cases in which the pressure of the air in the pleural cavity is the same as that of the atmosphere, the explanation appears more difficult, because it was thought that the elasticity of the lung alone was not capable of bringing about atelectasis. It is true that on opening a pleural cavity the lung collapses and still contains a large quantity of air, not only in the bronchi but undoubtedly also in the alveoli. It is certain, however, that if the animal survives the experiment by a few hours, the lung becomes completely airless.

It is not certain whether this is the result of absorption of the air

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* *Archiv für klin. Medicin*, vol. XVIII.

† Lichtheim, *Archiv für experimentelle Pathologie und Pharm.*, vol. X.

through the circulation, which still goes on, or whether the remaining elasticity requires a considerable period of time to effect the expulsion of the last remnant of air. According to Lichtheim's explanation, one is inclined to admit a cooperation of elasticity and absorption.* Elasticity alone is unable to render the lung completely airless, and needs the assistance of the circulation, as Lichtheim's experiments demonstrate. But this total atelectasis is comprehensible only on the assumption above referred to, that elasticity continues to act as long as a trace of air is contained in the alveoli.

A fourth subdivision of atelectasis would include the cases which, if Rommelaire † is right, develop after embolism of the pulmonary artery. But this occurrence is doubtful, and I am not prepared as yet to admit it. The weight of experimental evidence is decidedly against such a possibility.

ATELECTASIS IN THE NEW-BORN.

This form of atelectasis, also called congenital, occurs in debilitated, premature infants or infants apparently still-born after difficult labor.

As a rule, some mechanical obstacle is present in the respiratory passages; or the irritability of the respiratory centers may have been reduced by some injury to the brain, such as hydrocephalus or a hemorrhage during birth. Mechanical obstruction is most commonly due to the presence of meconium, which is drawn into the air-passages if the child prematurely engages in respiratory movements, either because placental respiration too early becomes insufficient or because respiration is induced by external stimuli before the head has been delivered.

In a certain proportion of cases the children are at first able to breathe quite well, but later the respiratory movements gradually become weaker and the lung returns to its primitive condition of atelectasis.‡

This question has often been discussed by medicolegal experts and obstetricians, and no one can doubt that the condition actually occurs. The air may disappear from the lung by absorption if the breathing is too weak.

A small focus does not always give rise to marked symptoms; in weakly children with a tendency to stupor and cyanosis atelectasis should always be looked for. On percussion dulness is usually found in such cases, most pronounced in the inferoposterior portions of both lungs. The dulness is not very intense; often the note is somewhat tympanitic. The respiratory murmur is weakened or altogether inaudible; but if the atelectatic focus is extensive, and the bronchi are clear, bronchial breathing may also be heard. In many cases a stenotic type of breathing is well marked; the yielding portions of the thorax, the infraclavicular fossa and intercostal spaces, retract during inspiration and the peripneumonic furrow becomes very distinct. The dyspnea is inspiratory in type.

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* *Loc. cit.*, p. 90.

† *Gaz. des hôpitaux*, 1883, p. 755.

‡ Pincus, *Vierteljahrsschr. für gerichtliche Medicin*, vol. XVIII, 1873; Winter, *ibidem*, 1887; Seydel, 1891; Klein, *ibidem*, 1892; Schröder, *Deutsches Archiv für klin. Medicin*, VI; Ermann, *Virchow's Archiv*, vol. LXVI.

Cough is frequently absent; if it exists, it depends on some concomitant condition. The degree of *cyanosis* present depends on the extent of the process. There is no fever.

The symptoms depend altogether on the extent of the atelectatic area; every gradation occurs, from cases in which nothing at all is found, to cases which give all the signs of a well-marked lobar pneumonia.

In view of the well-known variations observed in the pulse of normal infants, the statements in regard to retardation and acceleration of the pulse in atelectasis are to be received with great caution. It has never been determined that there is anything characteristic about the frequency of the pulse in asphyxia or atelectasis; the force of the pulse corresponds approximately to the other vital phenomena.

After a time the condition undergoes a change for better or worse: in the former case the symptoms subside; in the latter, the cyanosis increases; the pulse becomes small and rapid, the right heart engorged, the liver enormously congested, edema and urinary symptoms pointing to congestion make their appearance. Death is usually brought about by an intercurrent pneumonia, but even a simple atelectasis may increase to the point of asphyxia, and thus terminate life. Some infants survive the disease; but permanent injuries remain, the affected portions of the lung become atrophic, and the healthy portions hypertrophic.

Francke's case is typical of this condition.* Extensive areas of atelectasis in both lower lobes had evidently developed during or immediately after birth. The individual had attained the age of fifty-eight years. From the description of the microscopic findings it appears that extensive portions of the lungs had completely retained their fetal character. The condition of the thorax was regarded by Francke as characteristic of this form of atelectasis of both lower lobes. The deformity consisted in great bulging of the upper portions, with considerable increase in the vertical diameter. Below the sixth rib the circumference of the thorax was greatly diminished, as if the body had been constricted (wasp waist), and the lower edges of the thorax, finally, were bent over and outward. I do not believe that this condition of the thorax is as characteristic as Francke appears to think, since it is often found after rachitis. In this case the bronchiectasis and the compensatory emphysema are to be noted; both conditions occur in atelectasis as pseudo-compensatory phenomena. Hypertrophy, which is so characteristic of genuine fetal atelectasis, was not marked in this case. On

* *Deutsches Archiv für klin. Medicin*, vol. LII.

the other hand, contractions and adhesions were present, and must be referred to morbid conditions occurring in later life. The patulency of the foramen ovale and the dilatation of the pulmonary vessels were typically developed.

The **complications** of atelectasis are:

1. *Patulence of the foramen ovale and ductus arteriosus.* In atelectasis the aspirating and expansile power of the lungs, which in the normal state are so important for the onward movement of the blood, are considerably diminished; the stoppage of blood in the right ventricle is thus facilitated, and the latter therefore keeps the fetal channels open by utilizing them more and more to empty its contents. Closure of these channels takes place normally within two weeks after birth, and any circulatory disturbances occurring during this time interfere with this closure. In Francke's case the foramen only had remained patulous.
2. *Thrombosis.* This condition is also evidently connected with the disturbance of the circulation. It occurs most commonly and is most significant in the sinuses of the brain. These will be discussed more particularly elsewhere. Among other veins the renal are especially mentioned.
3. *Compensatory emphysema.* This has been treated at length in another section.
4. *Dilatation and hypertrophy of the right heart.* This is due to the same causes as in cases of emphysema.
5. In the atelectatic portions of the lungs *chronic progressive diseases* and their sequels—adhesions, bronchiectasis, phthisis—develop.

Diagnosis.—Atelectasis is determined from the history, the cyanotic appearance, the signs of interference with the respiration in circumscribed portions of the lungs, and the presence of dulness, which may, however, be very indistinct. Tactile fremitus is weakened or absent. This is a useful sign in excluding consolidation. The respiratory murmur is either very weak or even bronchial; after some respiratory stimulus it not infrequently rapidly approaches the normal. The intercostal spaces are prominent. The disease regularly involves the lower lobes.

Treatment.—In the new-born the mouth and throat are first of all to be thoroughly cleansed. A moist piece of linen is wrapped about the finger, and the cavities of the mouth and throat are gently wiped out, choking and vomiting being incidentally induced. Next, Schultze's method of artificial respiration should be resorted to, with cold affusions either directly or in a warm bath. Cold applied to the back and nape of the neck is the most powerful stimulant to inspiration. After a time the breathing can be favorably influenced with the aid of the faradic current.

Emetics are often recommended. Hertz, for example, is quite enthusiastic. (For small children syrup of ipecac with distilled water, of each 45 (f℥jss); one teaspoonful every hour. Or wine of antimony

5 (say, ʒj), water 75 (f℥iiss), syrup of ipecac 7.5 (fʒij); one teaspoonful every two hours).

I must confess that I consider them superfluous if the measures above detailed are properly carried out. In mild cases emetics are not necessary, and in severe cases they do no good. Nor do I recommend the employment of insufflation; the danger of producing interlobular emphysema is quite considerable, and blood and mucus are often driven into the alveoli, thus favoring the development of bronchopneumonia. The practice of anointing the skin with a stimulating substance, such as turpentine emulsion, seems to me less objectionable (oil of turpentine 30 (f℥j), water 60 (f℥ij), the yolk of one egg, made up in an emulsion).

Later on it is important to see that the child gets plenty of fresh air and is properly nourished; tonics and stimulants, such as quinin, cod-liver oil, malt extract, and small doses of wine, may be given according to the indications. If the children are cyanotic and stuporous, it is often advisable to resort to rectal alimentation, because such children often die of inspiration pneumonia when their death is erroneously attributed to atelectasis. Children can easily be fed with nutritive enemas; the simplest, and one that can be prepared at any time, consists of an egg with 10 tablespoonfuls of milk and 10 tablespoonfuls of water, to which is added a good pinch of salt.

ATELECTASIS ACQUIRED IN LATER LIFE.

I do not use the term atelectasis of adults, for it is impossible to draw a sharp distinction. In debilitated and reduced individuals of any age, often also in children, atelectasis may develop as the result of certain injuries. The clinical picture produced may vary greatly according to circumstances. During youth rachitis and diarrhea are the diseases that most commonly produce such a debilitated condition; next in order are the numerous infectious diseases, which also preferably occur during childhood. Chief among the latter are measles and typhoid fever; then follow croup, whooping-cough, scarlet fever, acute rheumatism, etc.

In these patients atelectasis regularly assumes the form of so-called hypostasis; that is to say, the inferoposterior portions of both lungs, on the right somewhat more than on the left side, become airless, or at least practically airless, and usually at the same time hyperemic.

Another form that belongs to this group is the atelectasis of kyphoscoliosis. In this form the atelectatic area is regularly found opposite the cleft-like posterior portion of the thorax on the side corresponding to the convexity of the curve.

Complete occlusion of a bronchus by a tumor or some other pathologic condition is followed by atelectasis which corresponds exactly in extent to the area supplied by the occluded bronchus. Here belongs, for example, the case reported by Cockle, in which the upper lobe of the right lung had alone become atelectatic as the result of compression of the bronchus by swollen lymph glands.* The same effect can no doubt be produced by masses of tenacious sputum, and it is not uncommon to see in the capillary bronchitis of infants and old men, if the disease becomes protracted, the coexistence of atelectasis and emphysema in different portions of the lung: atelectatic areas corresponding to bronchi that have become completely occluded, and emphysematous areas where there is only obstruction to the passage of air.

The literature contains one or two other communications on the subject of atelectasis the significance of which appears to me to be doubtful. The opinions advanced by the authors challenge criticism, for they appear to me to have been misled by erroneous speculation. I refer to the communications of Rommelaire and Levish.† In the cases which they describe extensive atelectasis developed acutely with fever, and an area of dulness over which the breathing was either absent or bronchial; vocal fremitus was abolished and there was expectoration of a peculiar "gummatous" sputum. It was thought repeatedly that a pleurisy existed, but exploratory puncture failed to reveal the presence of fluid. I think it is most probable that the authors had to deal with unusual forms of pneumonia; the occurrence of acute atelectasis as an independent disease has never been reported by any one else.

Abrams ‡ pointed out that circumscribed atelectasis is a physiologic condition that can frequently be demonstrated in persons with perfectly healthy lungs, and that circumscribed zones of dulness may be present at the apices or at the edges, or scattered over the entire lung, and disappear after a few deep respirations. The respiratory movements are somewhat limited, nothing is heard over the affected areas during quiet breathing; but on asking the patient to breathe deeply, a crepitation is produced. This condition is said to occur not only in debilitated, but also in perfectly vigorous persons, and to be followed by anemia.

Circumscribed atelectasis is no doubt a more or less common condition in debilitated individuals. In severe and protracted diseases, if the patient is asked to sit up and the lower portions of the lung are examined with a stethoscope while he takes a deep breath, a peculiar râle is heard which disappears after a few deep respirations (Lenhartz). It often happens in clinical work, in the examination of phthisical patients, that a change in the respiration heard over an apex disappears completely after it has been demonstrated to a number of students. The same thing is true of râles. These conditions may be termed pulmonary collapse, and should be distinguished from atelectasis, just as in emphysema it is found necessary to introduce a special subdivision under the name of pulmonary distention. The boundary-line between pulmonary collapse and atelectasis must, of course, always be arbitrary. Abrams' cases are evidently cases of collapse, and have no pathologic significance. If the condition is marked, it indicates debility on the part of the patient, and should not be neglected by the physician.

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* Hertz, in Ziemssen's "Handbuch," 2d ed., vol. v, p. 422.

† *Gaz. des hôpitaux*, 1883.

‡ *Klin. Centralblatt*, 1896.

To avoid repetition, I may be allowed to touch very lightly on the **symptoms** that may be produced by atelectasis in these various cases. It is essential that one of the etiologic factors that have been mentioned should be present, and that dulness and diminution in the respiratory murmur should be demonstrated, while at the same time the presence of an exudate or of infiltration is very improbable. The fine râle ("*Knattergeräusch*") that is made to disappear by vigorous inspiration is said to be particularly valuable; it is often very similar to a crepitant râle. The changes in the physical signs in different positions of the body and in deep respiration are often useful in doubtful cases, although there are many cases of atelectasis in which the physical signs are not affected by these factors, and which then lead to further sequels. The latter, in pronounced cases, consist in shrinking of the lungs with the formation of bronchiectasis and compensatory emphysema.

Compensatory emphysema is a constant complication of atelectasis, when the latter lasts any length of time. It is followed later by *dilatation* and *hypertrophy of the walls of the right heart* and by *symptoms of stasis*, as in emphysema.

The **treatment** of these forms of atelectasis naturally differs in many respects from the treatment of the condition in the new-born. In acute disease and when the patient is much reduced, prophylactic methods must be adopted to prevent the development of the condition. The good effects of cold bathing in the treatment of various forms of fever are partly due to their efficacy in combating atelectasis. Emetics were formerly recommended, just as later the pneumatic apparatus was lauded. For my part, I condemn the former, and substitute for the latter respiratory gymnastics, of which I have already spoken in connection with emphysema. Removal or mitigation of the causes, whenever possible, and constitutional treatment based on general hygienic principles are the weapons we must rely on in this battle, provided the basal disease does not frustrate all our efforts.

INFLAMMATIONS OF THE LUNGS.

BY

E. AUFRECHT, M.D.

INFLAMMATIONS OF THE LUNGS.

DIVISIONS OF INFLAMMATIONS OF THE LUNGS.

SINCE the beginning of the nineteenth century we have been able to differentiate, both anatomically and clinically, the inflammatory diseases of the lung substance from those of the pleura. The division of the various acute diseases of the pulmonary substance itself, however, has not as yet been finally settled.

The greatest hindrance to such a division is the anatomic conception of **lobar** and **lobular pulmonary inflammations.** Not that I am of the opinion that the anatomic difference is insufficient, or that a clinical or a bacteriologic and etiologic division would be a better one. Such a thought is foreign to my mind because I premise that in future we shall arrive at a point where our knowledge will be sufficient to differentiate the pathologic-anatomic differences of pulmonary inflammations just as well as we are able to do this in other diseases. The structural differences of the tissues will provide a certain method of differentiating these conditions, and especially will the causes of the affection give us definite knowledge. Every special cause of disease must produce an especial modification in the organic form of the malady. The pathologic-anatomic picture of the disease may be likened to the trunk of a tree, whose roots are the different causes, whose leaves and blossoms represent the various symptoms. Even the root of a particular plant variety may be readily differentiated from that of any other form.

It is, of course, easy enough to understand that the differences in the structural changes in pulmonary inflammations *were* sought and, as it was supposed, found in the grosser forms,—lobar and lobular disease of the lung tissue. The consequence of this false premise was the non-agreement of the anatomic findings and the etiologic demeanor. The lobar variety was made to include all diseases in which one or more lobes of the lung were affected *in toto.* These were counted among the so-called genuine pneumonias, and were grouped with that variety which even to-day is best known by the term "croupous pneumonia"; although the latter shows a characteristic anatomic lesion. I mean the evenly distributed granular appearance of the cut surface, which is absent in other pneumonias, in which an entire lobe is involved.

Instead of inquiring into the anatomic differences or the etiologic condition, on the one hand, or, on the other hand, instead of inquiring into the clinical phenomena, these different diseases were thrown into a common mass and designated lobar pulmonary inflammations; and from this there arose a truly kaleidoscopic description of the etiology and clinical symptoms. Infectious varieties, house epidemics, prison epidemics, transmission from birds to man, from cattle to man—everything was counted as belonging to this common group. If the subject is examined more thoroughly, however, one is easily convinced from the description of the anatomic findings that in the majority of these observations no simple variety of croupous pneumonia was present at all.

As this circumstance was not taken note of, the improper way was proceeded upon still further. Because lobar conditions in pulmonary inflammations did not quite come up to the anatomic characters of croupous pneumonia in all instances, and showed themselves to be directly transmissible in some, so croupous pneumonia was supposed to be a directly transmissible disease. This result seemed all the more likely because bacteria were found as the exciting cause of croupous pneumonia, and these were counted quite properly as the usual exciting element of the affection.

Croupous Pneumonia.—If we desire to arrive at a clear conception in reference to the etiology and the clinical course of the disease, we must, in the first place, recognize croupous pneumonia as an anatomically clearly defined variety of disease, strictly different from other varieties of lobar pneumonia.

In this way in one direction we may find a certain difference upon an anatomic basis, and the possibility is also near of dividing the various pulmonary inflammations in another manner.

Catarrhal Pneumonia.—The conception "lobular pneumonia" and its identification with catarrhal pneumonia is also not feasible if one wishes to lay stress upon etiologic and clinical conditions. As an example, the pneumonia following measles may be mentioned. This may arise as a lobar or a lobular inflammation, but never otherwise than as the result of a catarrhal inflammation of the finest bronchioles. For this reason it is well to abandon altogether the designation "lobular" as a definition of disease, and to retain only the term "catarrhal pneumonia."

It depends, then, upon the decision of the question which lobar inflammations are to be counted as belonging to the catarrhal variety, and if we have any criteria for this choice; and this, indeed, is the case. To demonstrate this point especially, in explaining catarrhal pneumonia, I shall mention but one characteristic condition, which is, however, in itself sufficient. This is the enormous inflammatory hyperemia of the smallest vessels and capillaries of the finest bronchioles, which postmortem are easily demonstrated in a microscopic preparation. The figures 6 and 7 in Plate 5 show this change plainly. Up to the present I have found this condition of the vessels in catarrhal pneumonia following measles, whooping-cough, and in those catarrhal pneumonias arising in scrofulous, cachectic children, and in influenza pneumonia.

The etiology of catarrhal pneumonia, however, is no longer so simple a matter as that of croupous pneumonia; the investigation later on will show a greater number of bacteriologic causes, whose effect will be noted in the anatomic lesions of the finest bronchioles, even if the cause of the affection is not due to a mixed infection. The latter condition I regard as unlikely.

Atypical Pneumonia.—Even with this the subject of the acute varieties of pneumonia is by no means exhausted. Besides the typical varieties of croupous inflammation, others are noted in which the etiologic designation in a complete form is not possible, and on account of this there is the greatest uncertainty. But one thing may be positively asserted: the impossibility of placing these varieties among the forms of croupous pneumonia, or even of showing a similarity between the two. A certain pathologic-anatomic designation is impossible because the changes in the pulmonary structure are very different. A lobar affection is frequent; but of an even granular appearance, such as is found in croupous pneumonia, there are no signs. The structure is splenified and only shows a smooth surface. Not rarely the disease is noted in smaller areas, and then a part of these areas is seen as a granular cut surface similar to that occurring in croupous pneumonia. But all these anatomic changes may arise in different combinations, where several fatal cases may be referred to a similar cause (house epidemics, prison epidemics).

Hence clinical medicine has no certain foundation. The designation of these pneumonias fluctuates according to the presence of one or another important symptom. Bilious pneumonia, pneumo-typhus, asthenic pneumonia, infectious pneumonia, are the usual terms used to designate these conditions.

I advise calling these forms, in contradistinction to croupous and catarrhal pneumonias, **atypical pneumonias.**

According to this, we may reckon in the category of the "non-tubercular" pulmonary inflammations—including the various diffuse and focal diseases, whose placing in this category does not require a special motive—the following diseases of the lungs:

I. DIFFUSE DISEASES OF THE LUNGS.

1. Acute.

(*a*) Croupous pneumonia.
(*b*) Catarrhal pneumonia. (Up to this time also designated as lobular pneumonia.)
(*c*) Atypical pneumonia. (Up to the present also designated as infectious or bilious or asthenic pneumonia, or pneumo-typhus.)
(*d*) Hypostatic pneumonia.
(*e*) Deglutition pneumonia.
(*f*) Desquamative pneumonia.
(*g*) Syphilitic pneumonia.

2. CHRONIC.

(*a*) Dust inhalation diseases.
(*b*) Pulmonary induration. (*Pneumonia fibrosa chronica*, cirrhosis of the lung.)
(*c*) Carcinoma of the lung.

II. FOCAL DISEASES.

(*a*) Abscess.
(*b*) Gangrene.
(*c*) Embolism.
(*d*) Infarct.

The justification of separating deglutition pneumonia from catarrhal pneumonia in this division is found in the fact that deglutition pneumonia and catarrhal pneumonia have erroneously been supposed to belong together, a subject to which I shall call especial attention at another place.

CROUPOUS PNEUMONIA.

PATHOLOGIC ANATOMY.

UP to the beginning of the nineteenth century we did not possess a certain anatomic foundation for croupous inflammation of the lungs. The authors of former centuries, from the time of Hippocrates, designated the most varied pleural and pulmonary diseases—serous and purulent pleurisy, pneumonia, pulmonary infarct—with a subjective combination of symptoms, as pleurisy and peripneumonia.

There can be no special reason for going into a historical analysis of the older descriptions at this point. In the works of Grisolle and Jürgensen are splendid historical reports. It might only be added that, according to Curt Sprengel's report, one of the oldest authors, Diokles of Karystus, correctly differentiated pleurisy from peripneumonia, and showed the seat of the former to be in the pleura and that of the latter to be in the lung, whereas later authors mostly were under the influence of the idea that in those pulmonary diseases in which pain was present a pleurisy must exist, and to this the most attention should be paid, until it came about that pleurisy and peripneumonia were considered as the same morbid process.

Morbid Anatomy.—To Laennec we owe the foundation of the conception of pneumonia upon a correct anatomic and clinical basis. He differentiated three well-defined and easily noted stages of the disease: Engorgement, hepatization, and purulent infiltration. The term "engorgement" he chose following Bayle; the name "hepatization," according to his report, is derived from Loelius a fonte (Morgagni). Laennec in especial emphasizes the granular appearance of the cut surface of the lung in the last stage. This appearance, in his opinion, is due to the transformation of the vesicles of the lung into solid granules, in consequence of the thickening of the walls of these vesicles and a simultaneous infarction (*état d'infarctus*) of the same. In the third stage, that of purulent infiltration, the organ has the same consistency and the same granular appearance as in the second stage, but the color is pale yellow or straw colored. Gradually the granules coalesce so that they are evenly covered by a yellow, translucent, tenacious, purulent mass. Frequently the three stages may be noted side by side in the same lung, either abruptly divided or merging one into the other. Usually the disease begins in the lower lobe of the lung.

Andral coincided with the division of Laennec into three stages; he characterizes these as engorgement, red softening, and gray softening, with simple purulent infiltration or abscess formation. Especially noteworthy is the fact that, according to his opinion, the disease is due to an inflammation of the pulmonary vesicles, whose inner surface at first gives rise to a mucous, sanguineous, and later to a purulent fluid.

The later works of Chomel and Williams add nothing new which would be of value to our present conceptions.

Rokitansky also differentiates three stages—the inflammatory engorgement (*engouement*), hepatization (*ramollissement*, Andral), and purulent infiltration (*infiltration purulente*). The first stage is the result of the filling of the tissue with "the inflammatory metamorphosed blood mass." Then follows the secretion of a very sticky, tough, brownish-red fluid, and finally the real exudation arises, with which the second stage of the disease begins. The lung is then of a reddish dark-brown color, tough but friable, and the cut surface is granular. The granules, however, are not, as was formerly supposed, the result of the swelling of the walls of the pulmonary cells, but an inflammatory product deposited in the cavities of the pulmonary cells. From the condition of red hepatization the pneumonia goes through various transitory stages, in the course of which it becomes paler, and finally takes on a reddish-brown, reddish-gray, gray, and finally a yellowish color, which is therefore designated gray hepatization, to the third stage of the disease, purulent infiltration. The granular texture has then disappeared. If a piece of such a lung is freed from pus by careful pressure and washing, the pulmonary substance again takes on its spongy cellular texture.

The seat of the pneumonic process is in the walls of the air-cells —that is, in the mucous membrane of the lungs; the product of the process is deposited in the cavities of the pulmonary cells. The pneumonia is therefore a croupous process of the mucous membrane of the lungs, similar to a parenchymatous croup.

Very often there are found, according to Rokitansky, the three stages, and these in the various stages of transition from one into the other, side by side. Usually in the middle and in the depths of the inflammatory lobes, gray hepatization and purulent infiltration are noted; toward the periphery, the reddish-gray red hepatization; and beyond this the inflammatory engorgement; and, finally, in the surrounding tissues simple stasis and, very frequently, acute edema of various grades may be noted.

Stages.—This division of Rokitansky's has in the main remained; only red hepatization has been separated from gray hepatization as an especial stage, so that nowadays authors, with the rarest exceptions, divide the course of a typical croupous pneumonia into four stages: first, inflammatory engorgement; second, red hepatization; third, gray hepatization; fourth, purulent infiltration, or, more properly, resolution.

Only Stokes divided the course of pneumonia into five stages, into the description of which it is not necessary to go.

The justification of the division into four stages will be explained in the following.

Macroscopic Appearance.—The macroscopic appearance of the lung in the various stages of croupous pneumonia, according to the unanimous opinion of authors, and also according to my own observations, with very few exceptions is to be described as follows.

The *first stage* of croupous pneumonia, the inflammatory engorgement (*engouement*), as an independent condition belongs to the greatest rarities. Death must then have taken place in the first twenty-four to thirty-six hours of the disease. I myself have had occasion to note two cases of this kind. In both cases the entire right lung almost exclusively, excepting a very small part of the upper lobe, was in the first stage of inflammation. The clinical symptoms had been such as to lead one to expect the condition which was found postmortem. Etiologically I must emphasize at this place that in one of these two cases—the other was not examined in this respect—in the pulmonary substance there was found not Diplococcus pneumoniæ, but a bacillus that in appearance closely resembled Bacillus coli.

More frequently one may, as was pointed out by Laennec and Rokitansky, note in the periphery of red hepatized areas a remnant of the first stage, engorgement. From a combination of the findings of the macroscopic examination, it may be concluded that primarily the diseased part of the lung is filled with blood, which leads to a simultaneous lessening of the air capacity of the part affected.

Accordingly the lung does not collapse in the same degree as under normal circumstances; it retains its *shape* as if it were inflated. At the same time it is heavier than a healthy organ. Upon pressing the fingers upon the upper surface, the structure may be pressed in like an edematous extremity [pitting] (Hasse, p. 267). Upon pressure, the cut surface tears more readily than normal pulmonary structure, which shows a decided difference from pure hypostatic congestion. The quantity of blood which is noted upon pressure, in the state of engorgement, is not so great as in hypostatic congestion, very likely because the contractility of the pulmonary structure in the former instance is very much lessened.

Of especial note is the fact that in both cases of pneumonia observed by me in the first stage the diseased organ upon section showed a dark, nearly blue-black appearance, and only became more red upon exposure to the air. This condition allows the deduction that the blood stream coming through the pulmonary artery into the pulmonary capillaries, even in the first stage was not sufficiently oxygenated.

According to Hasse's account, Morgagni and other observers had already called attention to the occasional appearance of darker blue-black areas in the stage of engorgement.

In the *second stage*, therefore, the stage of red hepatization, the lung shows, upon section, an evenly distributed red appearance. It is completely devoid of air. Only pneumonias of the apex may show a difference in this respect; the tissue need not be entirely airless. The cause for this will be explained later on. Further, the tissue is friable; it tears readily upon pressure with the fingers upon the cut surfaces. Granules are prominently noted, which correspond to the overfilled alveoli. This *granular appearance* depends upon the circumstance that the contents of the alveoli, through the contractility of the elastic fibers which are distributed through the alveolar substance of the lung, are pressed out. The size of the granules corresponds exactly to the size of the alveoli. Accordingly in children they are very small (Hasse, Ziemssen) and may, when an emphysematous lung becomes the seat of a pneumonia, attain a very large size. A case of this kind is described by Hasse. The granulations in this case attained the size of a hempseed. Damaschino has taken exact measures. He says that the granulations in children have a diameter of 0.07 to 0.11 mm.; in adults, 0.13 to 0.17 mm.; and in the aged frequently 0.21 to 0.27 mm. I have assured myself that in adults the granules vary between 0.17 and 0.21 mm. These masses coincide with normal pulmonary alveoli in a condition of distention. According to the investigations of Rossignol, the alveoli, after distention and drying of the lungs, have a diameter of 0.2 mm.

Size.—The question as to whether a pneumonic infiltrated lobe of the lung is larger than a normal one has given rise to much discussion. It may, however, be stated that the size of the lung in the deepest inspiration would give the size of the increase in volume in case of a pneumonic affection (Rokitansky, vol. II, p. 86).

The previously mentioned size of the granules in the stage of hepatization also proves this. The granules are never larger than the distended alveoli.

This engorgement of the alveoli with masses of exudate until the lung corresponds in volume to a fully distended organ explains the appearance of the impress of the ribs in disease of the lower lobe, which Rokitansky curiously did not credit.

According to Ziemssen, the impress of the ribs is exclusively due to the fact that the ribs are pressed from the surface upon the thickened pulmonary lobe. He believes that this only arises through compression of the thorax during forcible expiration, and believes that intense cough is the cause of these furrows.

I cannot coincide with this opinion, but believe that the cause of these rib furrows is found in the fact that the infiltrated lung retains the inspiratory size of the normal lung even during expiration. In rendering the lumen of the thorax smaller during expiration through the combined activity of the descending diaphragm and the lowering of the ribs, the infiltrated lung is shoved up without being able to accommodate itself to the lessened size of the thoracic space. In consequence a part of the pulmonary structure is compressed against the intercostal spaces, which still allow space for deviation, whereas the ribs remain stationary. It does not then depend *sensu strictiori* upon costal furrow-like impressions, but upon intercostal rounding out of the infiltrated pulmonary structure. Probably such rounding out *in vivo* happens more frequently than can be demonstrated postmortem. After the cessation of breathing, therefore after the discontinuance of the expiratory inward pressure of the lung in the intercostal spaces, with the aid of the negative pressure in the pleural space, a flattening of the upper surface of the lung may occur.

The increase in **weight** through the pneumonic exudate has also repeatedly been demonstrated. According to Eichhorst, a left pneumonic lung weighed over four pounds more than the right lung, which was normal. The cases of Eichhorst generally show an average increase in weight of 887 grams (2 lb. 4 oz.). Homburger and Kussmaul have shown an average increase in weight of 810 grams (2 lb. 2 oz.); Lépine, one of 538 grams (1 lb. 5 oz.) (Finkler, p. 22).

Bollinger has in 100 cases of croupous pneumonia shown the weight of the exudate. In 47 cases the weight was over 1000 grams (2 lb. 8 oz.) (of these, 8 were over 1500; maximum, 1805); in 39 cases, 500 to 1000 grams (average, 718 grams); in 14 cases the weight was under 500 grams (the average being 367 grams).

The *third stage*, gray hepatization, in the last decades, according to the unanimous opinion of all authors, has been separated from the stage of red hepatization and has been described as a special stage. This separation may, according to the descriptions which will follow later, be justified by the microscopic examination. Consistency and granulation of the cut surface in the stage of gray hepatization are the same as those of the preceding stage; the organ, however, no longer looks red, but pale yellow or grayish-yellow. The individual granules are readily differentiated from each other. "They are more demarcated, larger and stand out more prominently, are easier to isolate and lift out, and are looser, owing to the fact that they are surrounded by a gelatinous fluid which has accumulated around the wall of the vesicle (alveoli)" (Rokitansky, vol. II, page 88). In the cut surface frequently fibrin plugs are noted oozing from the finer bronchi, which may be pulled out to a certain extent.

In some few cases I found the connective-tissue structure of the lung greatly diffused along the course of the larger bronchi. On account of its gray hyaline appearance it was in marked contrast to the whitish-yellow parenchymatous structure. As the bacteriologic condition was not determined, it is impossible to decide whether such pneumonic changes may be attributed to croupous pneumonia.

In the *fourth stage*, that of purulent infiltration or resolution, the tissue has finally attained the highest grade of softening, or, more correctly speaking, of decay. It is the final stage through which croupous pneumonia passes, and in the nature of things shows the general course of the affection (Rokitansky, vol. II, page 89). Whereas the appearance of the surface and the cut section coincide with the appearance of the stage of gray hepatization, the granules have entirely disappeared from the cut surface; the surface being thickly coated with a thin purulent covering; it has the appearance as if the contents of the alveoli had become confluent.

But few authors have dissented from the opinion that every typical pneumonia has to undergo the four stages just described. Whereas Bennet previously and quite properly declared that resolution in pneumonia is not possible before the exudate becomes softened, fluid, and changed to pus, Grisolle is of the opinion that a lung in the stage of gray hepatization never returns to the normal condition, and Buhl * ([39], page 35) says: "It may be readily understood that even if it does occasionally happen that the stage of gray hepatization terminates in resolution, this favorable result is very much rarer in the case of purulent infiltration. Nevertheless, should this happen, the condition is entirely analogous to those cases of gray hepatization with delayed resolution."

The results of the microscopic examination, which will be later described, will prove the correctness of Bennet's view.

Only in pneumonias that run a very brief course of one to two days' duration does the disease not undergo all of these stages. Here a cessation of the process in the first stage must be presumed.

Histology.—A proof of the correctness of this view may easily be adduced, considering the diverse conceptions of the histologic processes in the first stages of pneumonia.

Only the microscopic examination of pneumonic lungs will allow us to judge of recovery in the individual stages as well as of the conditions which arise in the continuance of the disease from one stage to another.

The manuals and text-books in describing the beginning of the disease lay particular stress upon the condition of the circulatory system. So Förster remarks: "Microscopic examination shows almost complete filling of the capillaries with red blood-cells, filling of the vesicular structure of the lung with desquamated epithelial cells which have undergone fatty degeneration, and of epithelial cells which have become granular, as well as new connective-tissue cells." Rindfleisch says: "In the first stage all the vessels are completely filled with blood. A beginning exudation of an albuminous sticky fluid introduces the coming exudation and extravasation." According to Birch-Hirschfeld (p. 417), one finds "at the microscopic examination in the first stage that the capillaries of the alveoli are dilated and completely filled with blood; inside the alveoli, besides red blood-cells and exudative cells, swollen epithelium is noted."

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* The small figures throughout the text are references to the Literature on page 592.

After all that has been said, it must be stated that the general opinion is that the *first stage* of the inflammation consists in a *hyperemia of the capillaries of the lungs.* The accompanying conditions such as relate to the cellular elements, especially the epithelium, are regarded as a minor consideration or an accidental accompaniment of the process.

It dare certainly not be forgotten that the appearance of the hyperemia as a sign of the onset of the disease is handed down to us from a time in which it was not at all known that epithelium was contained in the pulmonary substance. Nowadays we are forced to take note of the actual existence of the alveolar epithelium, and are forced to give an especial examination to the part which it plays in this process.

Attention was previously called by Virchow, in his communications in reference to the blocking of the pulmonary arteries and its consequences, that the so-called inflammatory globules were nothing more than the vesicular structure of the lung filled with epithelial cells that had undergone fatty degeneration; that the vesicular structures of man and of mammals were surrounded by a basement epithelium which in the lungs of the human fetus he had found quite large, flat, completely smooth, translucent, usually with one, more rarely with two, very distinct, comparatively small, slightly granular nuclei ([243], p. 288).

Elenz and Eberth have, by means of the method of coating with silver, demonstrated a complete epithelial lining of the alveoli in the lungs of amphibians, and Colberg's researches have shown that the human lung, even after birth, retains its epithelium; that even in the alveolar structure of the lungs of adults a complete epithelial structure exists.

In complete accord with the before-mentioned authors, Schulze believes in the existence of a continuous but uneven epithelial lining of the alveolar structure of the lungs in the adult mammal; he, however, especially emphasizes that those with granular contents and a pale round nucleus, and with polygonal or unevenly round epithelial cells, are rarely met with or only in small groups, consisting of from two to four cells lying between large, pale, uneven, angular or slightly wavy, thin structureless plates, which have developed from the epithelial cells and their original structure, most likely through the pressure of the capillaries surrounding them and from the stretching of the distended alveoli; perhaps also, as Elenz notes, partly through blending of the neighboring epithelial cells.

Finally, Ranvier, likewise Küttner, confirm the existence of a complete alveolar epithelium. The latter proves that the original spindle form of the embryonic epithelium, which lines the entire bronchial tree with all its branches, becomes in extra-uterine life the basement epithelium of the alveoli, the cell adapting itself to the floor which it is to cover, and the nuclei becoming less discernible, following the spreading of the cell. In the lungs of dogs, after the injection of silver, the flat epithelial cell of the bronchioles proves to be 0.009 mm., the border of the infundibulum 0.013 mm., the basement cell of the alveolus which lies very near to it 0.05 mm., in size. Occasionally one notes in the alveoli of the infundibula one and the same basement cell spreading over the lining septum and with its halves lying in two neighboring alveoli.

Buhl's assertion[39] that the alveolar epithelium is not so much a continuation of the epithelium of the bronchi, as a lymphoid endothelial cell lining the inner surface of the alveolar wall, is not tenable, in view of the fact that the continuity of the epithelium from the hilus of the lung may be traced into the alveolar structure. [Pratt supports Mallory in the belief that the cells are endothelial. —Ed.]

According to Kölliker, human alveolar epithelium consists of a continuous layer of basement cells which show two entirely different elements: first, small cells with flat, polygonal, or round protoplasm, from 7 to 15 μ in size, which are found exclusively in the meshes of the capillary; and, second, larger manifold forms, apparently without nucleus, consisting of very fine plates of from 22 to 45 μ in diameter, which lie upon the blood-vessels. He, however, especially emphasizes that the epithelial plates may also extend into the meshes of the blood-vessels.

Feuerstack in the main coincides with Kölliker. Besides the small granular nucleated cells, he also notes larger nucleated hyaline plates, and, alongside of these, transitory forms of granular nucleated cells and plates without nuclei.

We may, therefore, take as a basis for our further investigations the fact that the pulmonary alveoli have a continued or continuous epithelium, and that the same contains two kinds of cells—namely, small nucleated cells, and larger ones, partly nucleated and partly plates without nuclei. Certain proofs of the correctness of this fact force themselves upon us in the investigation of the pathologic changes occurring in human lungs; the existence of two epithelial varieties is especially to be noted in inflammatory changes. From my investigations I am inclined to regard the non-nucleated plates as the result of a kind of desquamation of the alveolar epithelium, an analogous condition to the desquamation of epithelium in the epidermis. Let us first consider how far the epithelium has found recognition in the investigations of inflammations of the lung.

Beyer found in pneumonic lungs in which, macroscopically, the normal condition and transition stages from disease to healthy tissue, were found, besides the scarcely dilated capillary net, numerous cells, partly lying free in the alveoli, partly upon the inner surface of the plastered cells, which, judging from their condition, were obviously changed alveolar epithelium. They are similar to those found in the first stage of catarrhal pneumonia by Colberg; but they showed no multiplication of the nucleus. Agreeing with Wagner, he regards it as certain that the pus-corpuscles and fibrin met in croupous pneumonia go through the same metamorphosis as regards the development of the pulmonary epithelium, which Wagner found in the epithelium of the mucous membrane in croup and diphtheria. Heitler says that the epithelium in the beginning of the pneumonic process loosens itself; that the process therefore begins with a desquamation of the epithelium. In complete accord with Buhl, he declares that of these alveolar epithelia, and not only those in the stage of the beginning gray, but also those in the stage of red hepatization, large cells are found which originate in an endogenous cell formation.

I myself[4] was able to demonstrate a few years ago, upon the basis of my examination of pneumonic lungs, especially at the points of transformation of the disease into normal tissue, that at these points the alveolar epithelium was swollen, had a cloudy appearance, and for a large part was lying loosely in the alveolar lumen. From this I drew the deduction that this change demonstrated the first stage of the disease, and that only in connection with this affection of the epithelium,—with the superficial layer of the capillaries in the pulmonary alveoli,—either as the result of the inflammatory affection of the epithelium or because the same no longer gave a sufficiently tight cover, an alteration in the wall of the blood-vessels occurred which resulted in a hyperemia, an extravasation of blood, and, finally, in exudation. I declared that this view was all the more correct because the previously assumed hyperemia which was regarded as the beginning of the disease, with the subsequent serous exudation, could not at all be regarded as the signs of an inflammatory process. Hyperemia and serous transudation have as little connection with pneumonia as ascites has with peritonitis. Virchow[242] three decades previously had established the fallacy of Vogel's assumption that inflammation is capillary hyperemia plus *hydrops fibrinosus*. Unverricht alone declared that the loosening of the epithelium in pulmonary inflammation was an accidental factor, due to the structure of the organ, which had no essential significance in the inflammatory process.

Feuerstack, on the other hand, even regards the differences of structure in the alveolar epithelium as partly responsible for the morbid process. He declares that the change in the epithelium in pneumonia is inflammatory, but that it is not the primary process. The large hyaline flat cells hold themselves purely passive. They are partly torn from their base, and they partly remain as a cover for the alveoli. On the other hand, the nucleated granular epithelial cells react "actively upon the inflammatory hyperemia"; they become larger, and increase by proliferation. As a result of their increase in volume and number, and of the swelling of the capillaries in whose meshes they lie, they are pushed forward and floated away by the serum. Causative relations between the changes in the alveolar epithelium and the coagulation of the fibrinous exudate do not exist.

Finally it must be mentioned that Hanau returns to the view of Beyer, in that he believes that in pneumonia a coagulation necrosis of the epithelium *in situ* exists, and a croupous membrane formation due to the combined action of the necrotic epithelial cells and of the exudate from the blood course exists.

From these communications in reference to the anatomic behavior in pulmonary inflammation it appears that the alveolar epithelium plays a part which must not be lost sight of. Whether, however, it is to be regarded as the primary seat of the disease, for which view Heitler and I stand, cannot be regarded as definitely decided in view of contrary opinions held by some investigators. Other researches, therefore, must be taken into consideration; at first those in regard to the influence upon the pulmonary structure of the cutting of the vagus. Traube had proved that the pulmonary affection arising after a severing of the pneumogastric nerve was not due to the paralysis of the nerve-fibers leading to the lungs, nor to the narrowing of the glottis, but that it was due to the fluid which collects in the mouth and finds its way into the glottis, which is no longer able to prevent the passage of the same into the air-passages. Friedländer then conducted experiments upon rabbits by cutting the vagus and noting the changes occurring in the lungs; as a result of this he denies to the alveolar epithelium any active part in the inflammatory process. Swelling of the alveolar epithelium in the first stage of the inflammation by no means represents a specific morbid action or a specific sign of inflammation. This assumption is substantiated by the fact that large, round, swollen, alveolar, epithelial cells are produced not only in the first stage of inflammation, but also in edema of the lung, independent of the way in which the same was produced, and are also found in lungs that were previously filled with serum or any other watery fluid; even in lungs examined in serum, analogous changes arise. The usual views entertained: "There is no acute inflammation without the appearance of abnormal lymphoid cells in the affected parts"; and: "One has cause to entirely drop the parenchymatous inflammation," are probably not to be regarded as the results of investigations, but rather as the teachings of pathology existing during that period.

Dreschfeld arrives at different results. In the lungs of vagotomitized rabbits and guinea-pigs he found after four hours marked hyperemia with diminished air capacity, especially in the lower lobes of the lung. Microscopic examination showed the alveoli filled with large cells which contained four granules with shining nuclei. These cells were slightly granular, as were also the nuclei. Dreschfeld regards them as epithelial cells with beginning proliferation, and regards the process as of an inflammatory character. Simple imbibition with water or serum produces swelling in the cells, but without granulation of the protoplasm and without splitting of the granules. Ten to fourteen hours after cutting the vagus, if the animals are killed, they show in their lungs completely desquamated epithelium. In animals killed later (twelve to twenty-four hours after cutting the vagus) the alveoli are filled with large, swollen, granular epithelium containing several nuclei with small lymphoid cells and cells of medium size which have a hyaline nucleus. These latter cells are to be regarded as the daughter cells of the epithelium.

Frey, who gave careful consideration to former labors in this field, and especially to the experiments conducted by Traube, also made investigations in reference to the changes which occur in the lungs after cutting both vagi. He arrives at the conclusion previously given by Traube, that, as a result of the cutting, the inflammatory affection which arises in the lungs is but a consequence of the fluid from the mouth which flows into the respiratory tract. After unilateral cutting of the vagus very slight or no alteration in the lungs occurs. Any influence of the vagus upon the bronchial or vascular muscular structure of the lung he entirely denies. The diseased process is characterized by a serous effusion into the air-passages, hyperemia, consolidation, bronchopneumonic areas (especially composed of pus-corpuscles), vesicular and vicarious emphysema. The alveolar epithelium in these processes, which closely resemble the pulmonary inflammations in children described by Colberg, takes a passive part, showing opaqueness, swelling, and desquamation of markedly granular cells.

So, then, the results attained by cutting of the vagus show no uniformity of views. Nevertheless the conclusions of Dreschfeld are probably correct, and the changes in the epithelium must be regarded as inflammatory. The beginning nuclear increase in the alveolar epithelium and the later development of lymphoid cells can scarcely be regarded otherwise. No one has taken exception to these facts.

A further method to clear this condition is given by the experiments in which fluids of a more or less irritating character are injected into the pulmonary alveoli by way of the trachea.

Sommerbrodt noted after the influx of blood into the pulmonary alveoli the alveolar epithelium change through swelling, opaqueness, increase in the size of the nuclei, and increase in cell capacity, and develop into the so-called "large (giant) cells." Some of these cells had even taken up red blood-corpuscles; and here and there an increase could be noted, due to the segmentation of the nucleus. The blood, therefore, gave rise to a catarrhal pneumonia. In previously healthy animals experimented upon this process always resulted in complete recovery.

Through injection of liquor ferri (8 drops in 30 c.c. of distilled water) areas arose which were of the characteristic croupous pneumonic form; the alveoli were closely filled with blood-cells, with rich lymphoid (pus) cells, with detritus, and stringy, coagulable exudate.

Veraguth arrived at results which are worthy of close attention. He first demonstrated, in complete accord with Elenz, Eberth, Schulze, Colberg, Kölliker, through his own investigations upon rabbits, that the alveolar epithelium is a mixed one; that is to say, consisting of nucleated, small, round, polygonal cells and large, irregular, structureless plates, without nuclei. In the terminal alveoli the first are rare, not more than two being found in a group; the nearer the bronchial tube lies to the alveolus, the smaller the plates become, and the more frequently may be noted remains of the original nucleus, and the larger and more frequent are the nests of the smaller nucleated cells, especially in the depths of the alveoli. After injections of weak nitrate of silver solutions into the trachea of rabbits, there result hyperemia and serous infiltration of the parenchyma, and changes in the alveolar epithelium. The nucleated round polygonal cells of the same swell to large round masses, their nucleus is divided, their protoplasm becomes granular, opaque; a large part loosens itself from the cell stratum, and is found lying free in the alveolus (after six hours). The plates which are not nucleated do not swell, but take on a granular appearance, and soon degenerate into fine granular masses or masses of granular or smooth fibrillæ, which gather themselves around the round swollen epithelium and, together, loosely fill the alveolar structure (after from twelve to twenty hours). In the mean time the white blood-corpuscles accumulate in the dilated vessels and exude into the surrounding connective tissue and into the alveoli (after from twenty to twenty-four hours). In some parts of the lung the infiltrate is thick, small-celled, as in bronchopneumonia. In other parts it is lobular, or it forms, distributed over a large part of the lobe, a thick fibrinous exudate which in its entire appearance reminds one very much of croupous pneumonia (in from twenty-four to thirty-six hours after the injection). The decay of the non-nucleated plates of the alveolar epithelium is the primary condition for the development of a fibrinous exudate, but by no means the only cause of the croupous exudate, as Wagner and Beyer suppose. There develops but a preliminary and sparse meshwork which is not so smooth and shining as that of the exudate which arises afterward, which in the main appears to be a product of the elements of the blood.

In contradistinction to the views of Friedländer, Veraguth declares that the changes produced by nitrate of silver in the nucleated epithelial cells are the result of inflammatory processes, and believes himself justified in a pathologic-anatomic sense to bring up the question whether the agent (infectious or other) which in man produces croupous pneumonia, does not primarily and in the first place, and in the same or in a similar manner, act upon the non-nucleated plates of the alveolar epithelium, as he has seen in his experiments.

Gluzinsky also has demonstrated in an experimental way, that normal blood flowing into healthy lungs produces, even after twenty-four hours, a reaction on the part of the parenchyma of the lung, which in the first few days manifests itself by a desquamation of the alveolar epithelium of the lungs, and of the epithelium of the finest bronchioles, as well as by migration of the lymph cells, sometimes with consecutive peribronchitis.

The experiments here mentioned, in which fluids are brought in contact with the alveolar lumen, are in the first place of special value on account of the uniformity of the results. They show that the alveolar epithelium in all cases is damaged, and that an active change is produced which we must regard as inflammatory; an increase in the nuclei of the alveolar epithelium involves such a condition, as might be universally admitted.

Also the similarity with the changes in croupous pneumonia of man is especially emphasized. Following the swelling of the alveolar epithelium with increase in their nucleus, the exudation of red blood-cells and lymphoid cells (white blood-corpuscles) into the alveolar lumen takes place. There is no reason for believing in a primary action of the capillary vessels which line the alveolar walls.

So far everything is against the supposition that in man the beginning of a croupous pneumonia consists in a hyperemia of the capillary vessels. We are actually forced to concede to this process only a secondary part.

My own microscopic examinations of the primary stages in human pneumonia, conducted during the last year, give me the right to mention processes which coincide with the results obtained in animal experiments, and to substantiate my former opinion in reference to the primary inflammatory reaction of the alveolar epithelium. I had an opportunity to examine two cases of pneumonia in which death took place twenty-four hours and thirty hours respectively after the onset of the disease. The conspicuous dark-blue appearance of these lungs upon the cut surface I have already previously noted. The microscopic examination showed that the most marked, or, more correctly, the only changes, had taken place in the alveoli. The latter showed themselves almost exclusively filled with cellular material. Large round cells with large nuclei lay rolled together in the shape of a ball in the lumen of the alveoli (compare Plate 4, Fig. 1).

These cells are nothing but swollen alveolar epithelium. Their size alone makes their differentiation from white blood-corpuscles, which might come into question, an easy one. Besides, staining with Biondi-Heidenhain three-color mixture,* which I used almost exclusively in my investigations, shows that the protoplasm of these cells has a characteristic rusty brown appearance, which makes it

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* Pieces of the diseased lung about the size of a walnut were kept about eight days in a 5% solution of bichromate of potassium, which was frequently changed; they were then hardened in alcohol. After this particles of the thickness of several millimeters were fastened upon cork with fish glue, kept for from one to two days in absolute alcohol, and then finished and sections cut with the microtome. These were laid for from ten to fifteen minutes in undiluted Biondi-Heidenhain three-

EXPLANATION OF PLATES 4 AND 5.

FIG. 1.—Contents of an alveolus in pneumonia during the stage of engorgement. The alveolar epithelium, consisting of cells with and without nuclei, is granular and turbid; between the cells are red blood-corpuscles and masses of granular material, probably derived from the epithelial cells.

FIG. 2.—Multinuclear alveolar cells, formed by the coalescence of normal epithelial cells.

FIG. 3.—Three alveoli in croupous pneumonia during the stage of fibrin-exudation from the vessels. The alveolar epithelium is completely surrounded by threads of fibrin, which also penetrate the wall of the adjoining alveolus. The partial separation of the entire epithelial covering of the alveolus is to be attributed to the hardening of the specimen. On the right is a blood-vessel distended with red and white blood-corpuscles.

FIG. 4.—Part of an alveolus in the stage of red hepatization in croupous pneumonia. The capillaries are completely filled with blood. Cells of alveolar epithelium, with and without nuclei, are seen in immediate contact with the alveolar wall; except for these, the contents consist exclusively of red blood-corpuscles.

FIG. 5.—Part of an alveolus in the stage of gray hepatization. The large cells, with large nuclei, that form the alveolar epithelium lie next to the wall; the rest of the alveolus contains white blood-corpuscles embedded in a fibrinous network of regular structure.

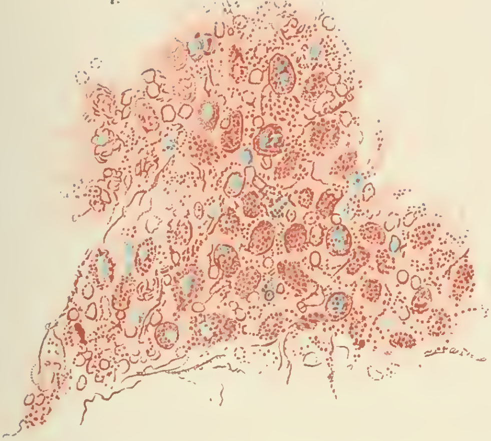
FIG. 6.—One half of the wall of a secondary bronchus in catarrhal pneumonia. The epithelium is filled with red blood-corpuscles. The vessels of the bronchial wall are distended with red blood-cells.

FIG. 7.—One half of the wall of a bronchiole, seen in oblique section, from a case of catarrhal pneumonia following measles. The lumen is completely filled with red and white blood-cells. The capillaries of the wall are practically injected with blood.

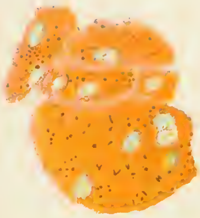
The specimens are all stained with Biondi-Heidenhain's mixture, except Fig. 5, which is stained with fuchsin (0.01 fuchsin-rubin in 100.0 of distilled water).

PLATE 4.

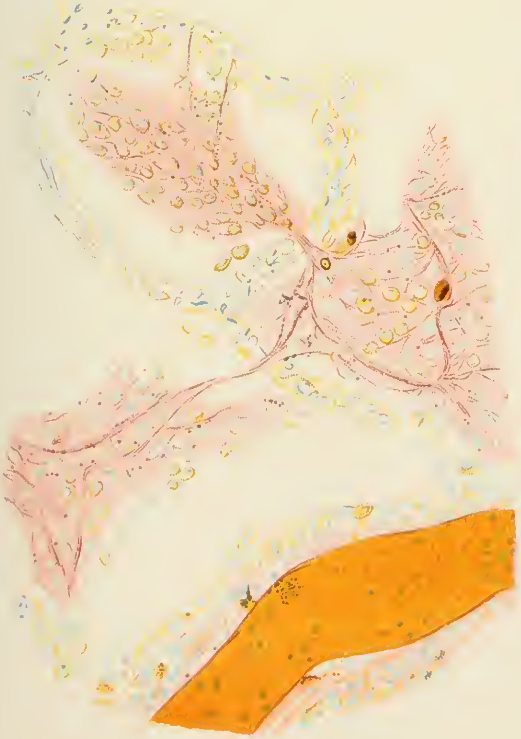
1.



2.



3.



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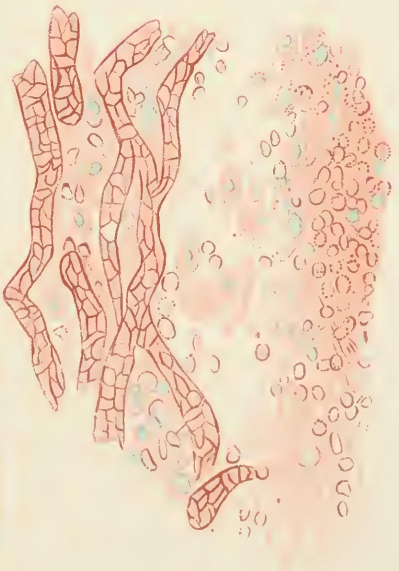
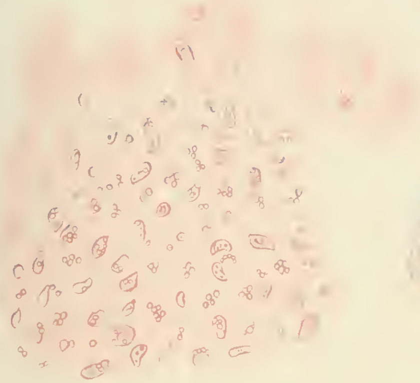


PLATE 5.

5



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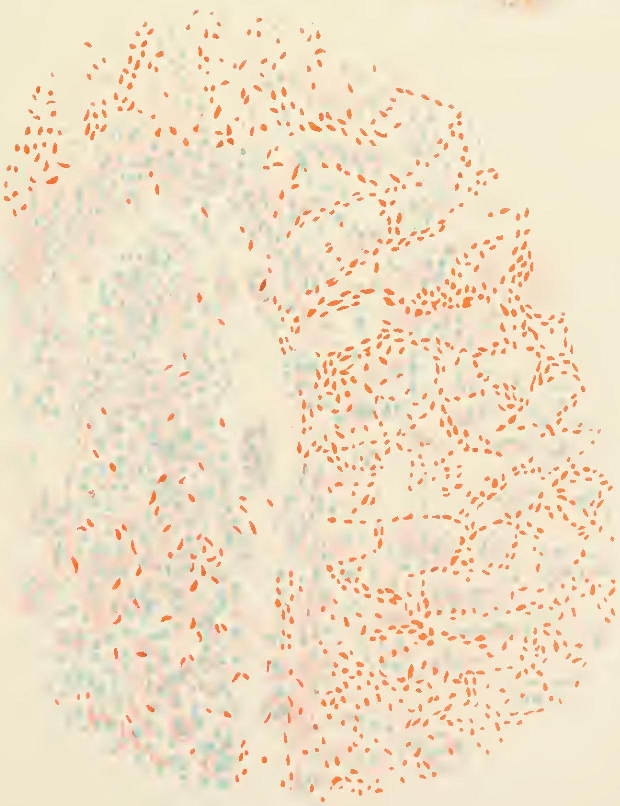

possible absolutely to differentiate white blood-corpuscles and the rest of the cells of the lung structure. [Pratt found in this transitional leucocytes, non-granular, many having phagocytic properties, and probably derived from the alveoli. Red blood-corpuscles and lymphocytes were also present.—Ed.]

Another part of the alveoli showed these large cells surrounded by fine, crumbling looking masses, which here and there inclosed the cells in the form of a ring. Besides this there are numerous red blood-corpuscles close to and between the swollen alveolar epithelium (compare Plate 4, Fig. 1).

A closely analogous condition of the alveolar epithelium I found in a case in which, to a pneumonia which ended fatally, a *myocarditis fibrosa chronica* was added which had nearly involved one-half of the left ventricle and had led to all the symptoms common to cardiac insufficiency. Only, in this case, among the swollen alveolar epithelium conspicuous large round cells were found with a greater number of nuclei.

Such conditions were found by Buhl[38] in the stage of transition from red to gray hepatization. He described them as large cells reaching in size up to 0.05 mm. in diameter, which are filled with globules which had attained the medium size of blood-corpuscles, and which he regards as mother cells, which have arisen from the basement-forming epithelium of the alveoli of the lungs. As, however, in some of these cells a different condition was noted from that of corpuscles in the fact that they were seen to contain granular nuclei, and occasionally even "a redoubled" nucleus was seen, so, according to his opinion, the pus-corpuscles in the mother cells could not have arisen through proliferation of the nuclei, but through endogenous production of the contents of the cell or have arisen as a free cell production.

According to Volkmann and Steudener, the origin of cells containing several nuclei is similar to the occurrence of purulent inflammation in the mucous membranes which are covered with flat epithelial cells. This may be due to an invagination of pus-corpuscles in the epithelium. A glance at figure 2, Plate 4, is sufficient to disprove such an assumption. The large nuclei exclude at the very onset any participation of the white blood-corpuscles. According to my opinion, the only correct explanation is found in the fact that these large cells containing several nuclei have not been produced through a multiplication of nuclei, but from a coalescence of normal alveolar epithelium.

As the changes described up to the present regarding the epithelium are the only ones which correspond clinically to the stage of

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color mixture and then placed in alcohol, water, and again cleansed in alcohol until no more coloring-matter could be extracted. The section was then spread upon an object-glass which previously had been dipped in alcohol while still in the alcohol dish, and after being removed from the latter, flooded with xylol. The alcohol was then allowed to drain off by holding the object carrier at an angle, and the section was mounted in Canada balsam.

onset of pneumonia, the conclusion must be drawn that this alteration is the first pathologic-anatomic effect of the disease. The stage of engorgement corresponds to the affection of the alveolar epithelium, which I have in a former work[4] regarded as belonging to the province of parenchymatous inflammation.

The foregoing pathologic findings suffice to prove that the first stage of pneumonia consists in swelling of the epithelium and multiplication of the nuclei in some of the epithelial cells, and in the escape of a few blood-corpuscles into the alveoli, as may be directly seen in some portions of the section. In the subsequent course of the disease this is followed by the escape of fibrin from the blood-vessels. Figure 24 illustrates the process so well that an explanation is scarcely necessary. In the interior of the fibrin filaments the alveolar epithelium rests. That both are almost always situated at some distance from the periphery is exclusively the fault of hardening the preparation.

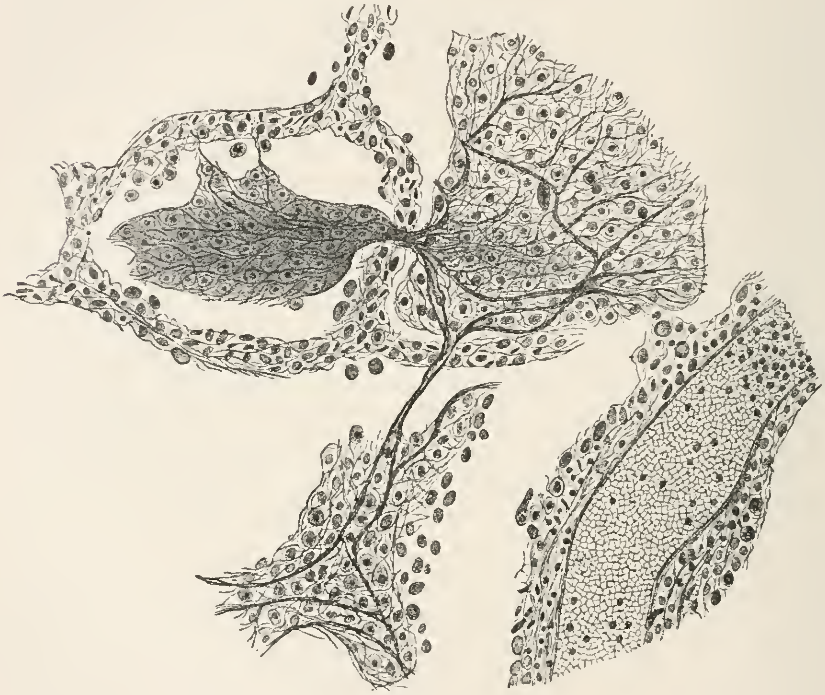

Fig. 24.

Hauser[96] formerly proved, in taking note of the researches of Wagner and Beyer, analogous conditions. He says that the alveoli are clothed as with a fibrinous pseudomembrane which in the hardened preparation is usually found more or less distinct from the walls of the alveoli, and gives the impression of tender, often manifold fibrinated filament which forms itself together in a network, giving the appearance of a tender membrane. Not rarely, also, the characteristic small rudimentary nuclei are noted, from which the delicate fibrin filaments or rows of fibrin granules stand off toward the periphery of the plate as very graceful stellate figures. The remainder of the cavity of the alveolus in the primary stage is still empty or contains serum, in which a few sparse cells are suspended. He, like Beyer, regards this fibrin net as the stage of onset of the pneumonic process, as a fibrinous degeneration of the alveolar epithelium.

A glance at the illustration (Fig. 24) suffices to exclude a degeneration of the alveolar epithelium. The epithelial cells in the interior of the fibrin filaments are retained *in toto*.

In another place Hauser[97] takes exception to the theory that the network of fibrin with which the alveoli are filled is produced solely by the coagulation of the inflammatory plasma, and that the threads of fibrin attach themselves to the alveolar wall. The fibrin threads, instead of being attached to the alveolar wall, as was supposed, pass through the wall at those points, and thus establish a communication between the fibrinous networks in adjacent alveoli. These communicating fibrin threads, according to Hauser, owe their origin to a coalescence of the hyaline epithelial plates, which have become embedded in minute, preformed, funnel-shaped canals (stomata) in the alveolar wall, with the hyaline plates of the adjacent alveolus. Through fibrinous degeneration of these plates the previously mentioned filaments of communication are formed. He, however, adds that these pore-canals have up to the present been found only in pathologic but not in normal lungs.

According to Hauser's opinion, then, croupous pneumonia of man is to be regarded as a typical croupous inflammation which is entirely analogous to the croupous inflammation occurring in mucous membranes. As in the latter, in addition to the exudative fibrin-production, the epithelium of the mucous membrane undergoes a fibrinous change, so also in croupous pneumonia the alveolar epithelium is first changed into a fibrinous pseudomembrane which lines the alveolus, after which the coagulation of the inflammatory exudate or infiltrate takes place.

Here then, also, the view is entertained that the affection arises primarily from the alveolar epithelium; the production of fibrin, however, is not sufficiently explained.

Ribbert substantiates the views of Kohn and Hauser in reference to the findings of strand-like fibrinous connections between the masses of exudate of the separate alveoli. He further demonstrates that some part of the alveolar plugs are richer in fibrin and other parts are richer in cellular elements. The alveoli which are especially filled with cells are found lying in groups, as in lobular pneumonia, and only coalesce later. The alveoli that contain more fibrin are affected later by the inflammation. The fibrin is found particularly upon the periphery of the alveolus, whereas the cells are found most copiously toward the center.

Ribbert's investigations were continued by Bezzola, who demonstrated the passage of the fibrin filaments through the alveolar walls. The explanation of the passage may be that at special places the epithelial coverings of two neighboring alveoli are placed directly one upon the other without the interposition of a capillary. If in such a case the epithelial layer upon both sides breaks down, the lumina of the alveoli are in free communication.

An explanation which is much more likely was given by Kohn for the fact that fibrin filaments emigrate through the walls of neighboring alveoli. The pores through which the fibrin filaments of several alveoli hold together are, according to his observation, tissue crevices, or tissue gaps, which are found between the capillary loops and elastic fibers of the alveolar wall. These, however, do not only form connections between the alveoli of one and the same alveolar passage, but between the alveoli of an entire lobule. One is, at least, able to follow these connective filaments between the fibrin nets of thirty or more alveoli. If these tissue spaces are not found open, it may be assumed that with the onset of the inflammation the epithelium desquamates, and in its desquamation the covers which are formed by the epithelium and which are found before each pore are lifted off. At the same time a loosening of a cement mass in the pores may occur, and in this way the passage may become free.

The findings of Kohn, Hauser, Ribbert, Betzola [and Pratt.—Ed.], in reference to the transudation of fibrin filaments through the alveolar wall I can completely substantiate, as may be noted from the illustration referred to above, as well as from figure 3, Plate 4. I am able, however, to add some few important conclusions. The latter are obvious to any one who has not a preformed opinion in reference to the production of fibrin from the changed epithelium. If the fibrin net, as has been illustrated in the figures and, as is always the case, contains alveolar epithelium, as well as cells and nucleated plates without nuclei in such large numbers that a destruction of the epithelium is not to be thought of; then, in view of the great number of the fibrin-threads, and their size at the points of exit from the alveolar walls especially, it is impossible to conceive how they could have originated in epithelium; we must, as a consequence, regard the blood as the only source of the fibrin which is found in the alveoli. There is, then, no need of the very forced explanation that the products of the fibrinous degeneration (coagulation necrosis?) of the epithelium of two adjacent alveoli coalesce as the result of degeneration, or separation from the alveolar wall, of non-nucleated epithelial plates belonging to two contiguous alveoli, which, at certain points on the alveolar wall, have become superimposed one upon the other; nor is it necessary to assume the presence of special stomata which do not exist normally. The transudation of the fibrin filaments through the alveolar wall is easily explained by the fact that the fibrin, which is produced from the blood, is primarily found in the neighborhood of the capillaries. As these capillaries wind through the alveolar wall, the fibrin must be found everywhere in the course of the capillaries, therefore in the wall as well as in the lumen of the alveolus. In fact it is most abundant in the tissues of the alveolar wall, where it meets with more resistance than in the lumen, and accordingly accumulates. Hence the strongest fibrin filaments are to be noted in this situation.

A further proof for the relations to the blood of the fibrin existing in the alveolar lumen and in the alveolar wall is found by Ribbert's researches in reference to coagulation of fibrin in the blood-vessels in pneumonia. I can substantiate these by a single finding.

It may, therefore, be asserted that the fibrin is not the result of degeneration of the epithelium, but arises exclusively from the blood, and follows the affection of the alveolar epithelium. The exudation of fibrin from the blood, however, involves disease of the capillary vessels, which shows itself in great capillary hyperemia.

That the latter is only a secondary process to disease of the epithelium is shown not only by all the previously mentioned anatomic proofs, but also by the fact that the diseased parts of the lung have an exceedingly dark appearance. This indicates insufficient oxidation of the blood in the pulmonary capillaries, and can only be explained by a primary deficiency of oxygen due to local conditions. This condition is found in disease of the alveolar epithelium. The parts of the lung which are not diseased, postmortem show a bright red appearance; the diseased parts postmortem, especially the cut surfaces, become light red only when exposed to the air.

The previously mentioned presence of fibrin in the lumen of the alveoli which accompanies the swelling, opaqueness, and increase in the nuclei of the alveolar epithelium, marks the transition to the second stage of the pulmonary inflammation.

In regard to the pathologic characters of the lung in this stage, I said,[4] in 1875: As I took advantage of the opportunity for the first time to examine microscopically a lung in the stage of red hepatization, I was surprised that the microscopic picture coincided so little with what I had learned to regard as being characteristic of this stage. I had expected to find in the mass filling the alveoli, which in this stage gives the diseased lung upon section the well-known granular appearance, an exudate *in optima forma.* I had expected to find, according to all our views of an exudate, the characteristic granulation, or pus-cells, the indifferent cells, which after the teachings of Cohnheim I regard as emigrated white blood-cells. But the objective findings in this, as in the remaining cases of red hepatization which I had an opportunity of examining later, were not in accord with my expectations. The contents of the alveoli consisted almost exclusively of red blood-corpuscles, whereas white corpuscles were only present in very small numbers, so that the proportion between the white and red blood-corpuscles was not greater than that found in the circulating blood. The blood-corpuscles were embedded in a dense but highly irregular net of fibrin filaments, which frequently terminated in bundles, and wherever they were visible in their entire extent, appeared long, stiff, and shiny—in short, like fibrin found in any ordinary blood-mass. Fibrin filaments and blood-corpuscles formed a dense mass which usually was found in the alveolar cavity, so that between every mass and the alveolar wall a small free space existed, traversed by a few fibrin filaments, which therefore connected the wall of the alveolus with its contents.

From these facts I drew the conclusion that the second stage, red hepatization, was not the consequence of an exudation, but only the result of the coagulation of blood, which arose from the torn capillaries of the alveolar wall. [Pratt supports Jürgensen in the observation that the blood is in the capillaries and not in the alveoli.—Ed.] In this connection I concurred with the report of Rindfleisch "that in a few, even in rare cases, the red blood-corpuscles were so massed that one might ask whether the exudate had not better be designated as an extravasation." In a similar manner Virchow ([244], p. 740) insists upon the distinctly hemorrhagic character of red hepatization. He says in reference to it: Genuine or legitimate pneumonia is characterized by the deposition of fibrinous products in the alveoli. The designation "croupous pneumonia" is really incorrect. The variety arising in croup belongs to catarrhal pneumonia. The usual pneumonia of adults differs from croup by the very important circumstance that the fibrin exudation is not pure, that the initial process, in fact, is hemorrhagic. Not only is the early sputum bloody, but the beginning hepatization is red; that is to say, bloody. The later fibrinous material is therefore not a pure product of the exudation, but it later becomes yellow, and only by and by takes on the so-called pure fibrinous character in the stage of actual (yellow) hepatization, in that the blood-corpuscles are absorbed and the hemoglobin is metamorphosed. For this reason Virchow for a number of years has used the term "fibrinous pneumonia" to designate the process which produces the actual (yellow) hepatization. If he does not call it "hemorrhagic pneumonia," it is owing to the fact that this name belongs to the metastatic form, which begins with hemorrhagic areas (*foyers apoplectiformes*, Cruveilhier).

It coincides with all the objective findings if I declare that the occurrence of a hemorrhage into the alveoli is characteristic of the second stage of pneumonia and as determining the stage of red hepatization. The reason for this condition unquestionably depends upon the circumstance that the capillaries of the alveoli are denuded of their protective epithelial covering, and in consequence molecular changes, which until then could not take place, occur in their walls, so that they tear under blood pressure and the contents of the capillaries, the blood, flows into the alveolar lumen in the direction of least resistance. The phenomenon depends upon the structure of the lung itself and is not identical with hemorrhagic inflammations occurring in other organs. Inflammations in other organs usually retain their hemorrhagic character permanently, whereas in pneumonia the hemorrhagic element typically and regularly disappears; in other organs the presence of hemorrhages implies a severe morbid process, while in pneumonia hemorrhage into the alveoli is found by experience to be a favorable prognostic sign. In those cases in which the hemorrhage does not give rise to complete filling of the alveoli, as in pneumonia of the upper lobe, in desquamative pneumonia, and in white syphilitic pneumonia, the course of the affection is less favorable, and a complete resolution is less certain or not to be expected at all.

Red hepatization can rather be compared to a thrombus formation in an injured blood-vessel. The contents of every alveolus represent a thrombus for the torn alveolar capillaries. Also the further progress, the transition of the red hepatization into gray hepatization, makes it justifiable to employ the analogy between this affection and the changes in the blood-vessel thrombosis.

After the changes just described in the alveolar epithelium, the transudation of fibrin into the alveoli, and the extravasation of blood from the lacerated capillaries in the alveolar walls have been completed, the process which has given rise to the designation "gray hepatization" becomes dominant. The characteristic feature of this stage is the almost complete filling of the alveoli with round cells, which, based upon the investigations of Cohnheim, must to-day be considered as emigrated blood-corpuscles. Certainly this emigration does not occur at once; the changes in the capillaries and the stasis of the blood in the same are the fundamental conditions for this emigration. The stasis, however, obviously occurs at the beginning of the changes in the alveolar epithelium, but the emigration itself obtains the upper hand only with the stage of gray hepatization; when the latter occurs, the contents of the alveoli become flooded with white blood-corpuscles.

Upon microscopic examination one finds in those areas where the alveoli are still filled with pure coagulated blood that the finest bronchial termini are occluded with white blood-corpuscles, just as if their function was to completely prevent the outer air from gaining access to the lumen of the alveoli. In other places white blood-corpuscles will be seen within the alveolar space between the alveolar wall and the mass consisting of fibrin and red blood-corpuscles, which is somewhat removed from the alveolar wall, so that this mass is practically enveloped by the corpuscles. Gradually the red blood-corpuscles entirely disappear, and only here and there can still be found small particles which seem to be the debris of the red cells. The network of fibrin threads, however, remains longer; it conceals the white blood-corpuscles after the latter have occupied the places where the red cells were previously situated. As a matter of course this substitution cannot be objectively traced, but that it does occur, must be as undoubted as the sequence of red and gray hepatization;

the one is as certain as the other. The first-appearing fibrin and that present during red hepatization seem only to disappear with the progress of the exudation or with the termination of the stage of gray hepatization; at least there is seen in gray hepatization, after the specimens have been hardened, only a fine network with meshes of almost equal size, and exactly corresponding to that seen in croupous membranes of the larynx (Wagner) and in wounds which heal by first intention.[3] This network, however, differs markedly from that seen in the precipitation of blood fibrin into red hepatization. It forms an evenly distributed network (compare Plate 5, Fig. 5), while the fibrin threads present during the stage of red hepatization are, so to speak, attached to the alveolar wall by a common trunk and thence separate in a radiating manner (compare Plate 4, Fig. 3). The origin of the fine, regularly arranged, small-meshed fibrin network which is present in the stage of gray hepatization can only be attributed in this stage of the disease to the blood. The fibrin escapes from the blood-vessels along with the white blood-corpuscles. The alveolar epithelium, however, does not suffer any further changes during the stages of red and gray hepatization; at least a portion of the same, as is seen in figures 3 and 4 of Plate 4, shows no other changes than those present in the beginning of the process; the epithelium always lies in the neighborhood of the alveolar wall. [Pratt observed polymorphonuclear leucocytes; large phagocytic cells, pigment-carrying cells, evidently derived from the cells lining the alveoli, and having in their inclusion, red blood-corpuscles, lymphocytes, and plasma cells; and desquamated epithelial cells.—Ed.]

From these microscopic findings the change in the appearance of the lung in the third stage is also easily explained as well as the gray or grayish-yellow color in contrast to the red of the second stage. There is no need of the hypothetic explanation "that, on account of the newly occurring infiltration, the blood-vessels are subjected to external pressure which influences the in and out flow of the blood" (Rindfleisch, p. 389). Still less is the gray hepatization, as Buhl supposes, "a phenomenon resulting in death of the parts which depends upon a lessened power of body and heart and the pathologic anatomy of which depends upon the fact that the extravasations become pale, the fatty degeneration adds its color, and the oligemia continues" ([39], p. 33). Neither is the phenomenon related to thrombosis in the vessels (Ribbert, p. 364). The gray color of the lung in this stage is to be looked upon simply as a substitution of white blood-corpuscles for the red. [Pratt believes it is due to empty capillaries only.—Ed.]

The fourth and last stage of croupous pneumonia is purulent liquefaction (resolution) of the exudate in the alveoli. Purulent liquefaction is the typical and necessary termination of every inflammation that goes on to gray hepatization; for without the liquefaction of the exudate coincident with suppuration, disappearance of the exudate could not take place. What chemic changes this phenomenon depends upon, has as yet not been made clear. We have, however, not gone any further than the view of Hasse, that the coagulated fibrin upon contact with pus becomes loosened and assumes a pale yellowish, sticky consistency. Microscopically, however, it will be seen, in preparations which macroscopically show the transition of a gray hepatization into purulent resolution, that the fibrin meshes have disintegrated into a number of correspondingly arranged granules, so that a granular disintegration of the fibrin renders a liquefaction of the exudate possible.

The complete disappearance of the exudate from the lung takes place principally by absorption. This view is not, it is true, substantiated by the histologic findings, which have so far failed to yield any positive proof of the occurrence of resolution. Only clinical observation justifies such a conclusion. Whoever attentively observes the infiltrated lung in pneumonia from the beginning of the crisis, and elicits the condition by percussion, cannot fail to observe that in certain cases within twenty-four and even twelve hours a pulmonary lobe completely infiltrated may again become entirely filled with air, while the accumulated expectoration is so little that even a most superficial estimate will show that the difference between the expectoration and infiltration is so marked as altogether to preclude the possibility of the infiltration having been expectorated.

After a description of the most important pathologic-anatomic feature of the pulmonary inflammation, the following is still to be remarked:

Bronchial Casts.—A frequent accompanying phenomenon of pneumonia is the finding of the so-called *fibrinous casts* in the finer and finest bronchi. Remak first described these in 1845. They represent branched cylinders with dichotomous divisions, thickened at the points of division. They consist of fibrin as well as of pus-cells, and contain also a few cylindric epithelia, which occasionally are still supplied with cilia. Remak found these principally between the fourth and the seventh days, and one, in an exceptional case, on the fourteenth day of pneumonia. In fifty examined cases they were present each time. Biermer missed this coagulation in 25 cases. Its presence corresponds to the stage of hepatization. He supports Remak's statement regarding the early appearance of this phenomenon. Only once did Biermer find bronchial casts in the expectoration and also the other symptoms of hepatization in a patient three weeks after the observed beginning of the pneumonia. He could only find the club-shaped processes which represent impressions of the alveoli twice during the disease, which shows that the bronchial coagulum is easily torn off from the alveolar contents. This thickening is more frequently observed in the cadaver when the fibrin coagulum is carefully drawn from the bronchi.

Damaschino says that this fibrin coagulum, which is an important sign of pneumonia and can be always demonstrated, possesses a peculiarity not yet called attention to, which facilitates the differentiation between this condition and the membranes of a diphtheritic bronchitis. The latter are tubular formations while the pneumonic coagulum does not possess any opening. This fact justifies the assumption that the coagula originate from the inflamed pulmonary vesicle. Furthermore, in bronchi of medium caliber the fibrin exudate covers the ciliated epithelium in a continuous layer. These formations, which represent the ramifications of finer bronchi, are frequently arborescent structures and do not occur only in adults; he states that he also found them in the pneumonic expectoration of a child eight years of age.

The view of Damaschino, that the fibrin coagulum in pneumonia represents solid material, is only applicable to the contents of the finer bronchi. If the fibrinous bronchial contents extend from the alveoli into the bronchi with a wider lumen, cylindric-formed exudates must also occur, just as they do in croup. Of course, this cannot be frequent, for only Lebert [143] reports a case in which a croupous membrane ("*des fausses membranes*") extended from the left hepatized lower lobe, from the finest bronchioles into the chief bronchus. In another place Lebert [144] refers to this case, and adds that the fibrinous cylinders had numerous branches and represented a bronchial twig with its various ramifications, that they had a milky white appearance, and that the larger cylinders, of the thickness of an ordinary writing-pen, were hollow, while the smallest ramifications did not show any canal-shaped lumen.

The microscopic examination shows that the principal constituent is a streaky, granular, thready material which embraces many pus-cells and but few epithelial cells. The fine stripes of the thready material are very plain, very fine, sharply outlined, and run partly parallel and partly divergent.

The origin of the fibrinous bronchial expectoration is not difficult to explain when we consider the processes described as occurring during gray hepatization; it represents a process in the finest bronchi similar to the emigration of the white blood-corpuscles into the alveoli, and histologically are the same as the alveolar contents which are inclosed in a uniform fibrin network, during gray hepatization. As has been remarked above, the finest bronchioles may be filled with white blood-corpuscles at their points of transition into the alveoli even in the stage of red hepatization; that is, while the alveoli are still filled with pure blood; from there the exudate may extend into the larger bronchi, and then form fibrinous masses.

Thrombosis.—A rarer occurrence in inflammation of the lungs is *venous thrombosis* occurring within the inflamed lobe, and still rarer is its extension beyond this lobe. Hasse says: In the third stage the circulation appears to be entirely interrupted in the affected portion of the lung; at least one finds that the smaller branches of the pulmonary artery, and occasionally also the pulmonary veins, are filled partly with coagulated blood and partly with fibrinous concretions.

Similarly, Virchow states[243] that obliteration of individual branches of the pulmonary artery frequently occurs in pneumonia. In this he sees the proof for the secondary coagulation of a column of blood impeded by mechanical interruption. I saw a marked pulmonary venous thrombosis in a case of fatal traumatic pneumonia. [Pratt found in the smaller vessels and capillaries in over half the cases, giant cells of bone-marrow, transition cells; plasma cells and phagocytes were found.—Ed.]

The occurrence of such a thrombosis may be traced to a coagulation in the smallest vessels as a consequence of the local stasis. Ribbert observed such a coagulation[202]; I also observed a pneumonia which became fatal during the third stage. The process is entirely analogous to venous thrombosis occurring in the leg originating from a small muscular vein. It is not justifiable to attribute the thrombosis to compression of the tissue by the exudate; no more is it justifiable to consider it due to an occlusion of the blood column caused by a mechanical interference.

Pleurisy.—The pleura corresponding to the affected lobe regularly becomes involved in the inflammatory process to a variable degree. In the mildest grades the pleura over the diseased lobe loses its glistening appearance and the blood-vessels become distended with blood. Punctate hemorrhages occur, and the surface of the membrane is often covered with delicate easily detachable deposits of fibrin. [Polymorphonuclear leucocytes, transition cells, and blood-corpuscles were caught in the fibrin meshwork.—Ed.] Quite frequently there are, in addition, large serous effusions containing numerous threads of fibrin, arranged in the form of a spider's web and extending from the visceral to the parietal layer of the pleura. More rarely the effusion consists altogether of pus and contains larger masses of fibrin, as large as a plum or even larger, scattered through the purulent fluid. In cases in which the empyema has been treated by operation, a coagulum of this kind may be large enough to occlude the opening in the thorax made by resecting the ribs.

A pleural effusion may also occur when the upper lobe only is involved; if the layers of the pleura are not adherent, the fluid will, of course, gravitate to the lower portion of the thorax. This association, which was first pointed out by Traube ([237], p. 16), is comparatively rare. The pleurisy in such cases corresponds in position to the diseased lobe; the fluid portion of the pleural exudate falls drop by drop into the lowest portion of the pleural sac, and compresses the lower lobe of the lung to a degree proportionate with the quantity of exudate present.

Bronchial Glands.—In direct relation to the pulmonary disease is also the occurrence of enlargement of the bronchial glands. Rilliet and Barthez observed this in children, and Grisolle observed it in adults. The latter once found a gland which, according to its appearance and consistency, was looked upon as representing purulent infiltration.

To avoid repetition, other rare complications affecting the lung tissue itself and the influence on the anatomic relations of other organs will be discussed in the section on complications. [The glands are involved late in the disease. They are distended with cells, fibrin, and coagulated serum.—Ed.]

THE CAUSE OF CROUPOUS PNEUMONIA.

Anticipating bacteriologic examinations, v. Jürgensen explained that: Croupous pneumonia is a general, and not a local disease. The inflammation of the lung is only the principal symptom; the clinical phenomenon cannot be explained by the local affection. It is necessary that we assume a specific cause. Croupous pneumonia belongs to the group of infectious diseases ([117], p. 143).

Klebs was the first one to demonstrate the presence of bacteria in the sputum of pneumonia. He called these bacteria "monadins." Later, Ebert and Koch also found "pneumonia cocci" in the inflamed lung, in the accompanying meningitic exudate, and in the blood. Leyden and Günther demonstrated these in a pneumonic lung during life by means of puncture.

We are indebted to Friedländer for an extensive investigation which first brought the question prominently into view. This is not the place to give an exact description of these preliminary labors which led to the study of the cocci found in pneumonia. The text-books on pathologic mycology, above all, that of Baumgarten, deal with the question in an extensive manner. Neither is this the place to consider the manner of cultivating the bacteria, no more than to consider the methods of auscultation and percussion when discussing the symptomatology.

Concerning Friedländer's pneumococcus, Baumgarten says: Friedländer's microbe is certainly only demonstrable by means of culture methods peculiar to its cultivation in a small portion of cases of typical croupous pneumonia; and even in these cases the fact is not to be ignored that the specific pathogenic microbe may have disappeared and a micro-organism, that has secondarily gained ingress, may have contributed to the production and further development of the pneumonic process. Neither have the results of experiments on animals given sufficient proof; for, irrespective of the fact that inflammatory changes in the lung, as they are caused by the pneumonia coccus of Friedländer, may also be caused by other bacteria as well as by chemical injuries, this pneumonia coccus proves itself entirely ineffectual in rabbits; why cannot man also be indifferent to the same? It is therefore questionable whether Friedländer's cocci have anything at all to do with the causation of genuine croupous pneumonia; and the theory, which until quite recently was generally accepted, that the Friedländer microbe is the cause, or at least one of the causes, of genuine croupous pneumonia in man, cannot now be regarded as sufficiently justified by the facts.

The observations of Fraenkel and Weichselbaum first permitted us to reach more certain conclusions concerning the nature of the exciting cause of pneumonia.

Fraenkel succeeded in cultivating from lungs affected by pneumonia under high temperature (most suitably at 35° C.—95° F.) an oval-shaped diplococcus whose shape unmistakably resembled that of a lancet. When transmitted to animals, this diplococcus showed itself markedly virulent. A small quantity of the culture injected under the skin of a rabbit caused death in from twenty-four to forty-eight hours, the blood being filled with characteristic diplococci. When the culture is injected through the wall of the thorax into the lung, there generally arises a pleurisy and fibrinous pericarditis; occasionally there also results a lobar inflammation of the lung which anatomically corresponds to croupous pneumonia in man. Similar diplococci have also been found in empyema following pneumonia and in meningitis complicating pneumonia.

Fraenkel considers it especially worthy of notice that this diplococcus rapidly loses its pathogenic properties both by cultivation at 41° to 42° C. (105.8° to 107.6° F.) and by an insufficient recultivation of the colonies.

Weichselbaum examined 129 cases of inflammation of the lung of various kinds and found: (1) A coccus provided with a capsule, lancet-shaped and occasionally round, which corresponded to Fraenkel's Diplococcus pneumoniæ, and which was generally arranged in pairs, although it also occurred singly in short chains of four, six, or more cocci; (2) the Streptococcus pneumoniæ, similar to the first, but frequently in chain formations; (3) the Bacillus pneumoniæ, which likewise posseses a capsule; (4) Streptococcus.

Among 129 cases he found Diplococcus pneumoniæ 94 times; and in these, 54 times in pure culture. Eighty of these were primary pneumonias and five secondary. In the diseased lungs were found Diplococcus pneumoniæ as well as Bacillus pneumoniæ; and the more recent the process, the more micro-organisms were found and the more pronounced was the capsule formation. In areas of gray as well as of red hepatization either a few non-encapsulated and poorly stained organisms were present or none at all were noted.

The bacteria of pneumonia, however, are also found in complicating pleurisy, pericarditis, peritonitis, and meningitis. In the latter disease an extension of the process could be traced along the cellular tissue of the neck to the submucosa of the pharynx and to the accessory cavities of the nose, and in all these places the presence of Diplococcus pneumoniæ could be demonstrated, so that it had to be accepted that a continuation of the process from the accessory cavities of the nose, and particularly from the labyrinth of the ethmoid bone to the lymph channels and thence to the meninges, gave rise to the meningitis. The possibility, however, should not be excluded that the exciting cause may also have reached the meninges through the blood.

Injections of cultures of Diplococcus pneumoniæ into the thoracic cavity of rabbits produced, in addition to pleurisy, pericarditis, and enlargement of the spleen, frequently also pneumonia in one or both lungs, but generally in the form of hepatization.

According to Weichselbaum, there is no specific pneumonic virus; genuine croupous pneumonia can also be caused by several varieties of fission fungi; it is a local disease.

But if we add to these observations that Wolff among 70 cases found the diplococcus of pneumonia in 66 cases, we are almost forced to acknowledge that the view of Fraenkel is correct—namely, that Diplococcus Fraenkel-Weichselbaum is the exclusive cause of croupous pneumonia. Concerning the few cases in which Weichselbaum could not demonstrate it, Baumgarten says: "If we consider that the method of cultivation adopted by Weichselbaum does not exclude a possibility of overlooking the diplococcus; further, if we remember the extraordinarily rapid disappearance of the vitality of the coccus in artificial culture-media, which allows of the analogy of a similar behavior also for the natural cultures in the infected organism, the small number of failures in the slight percentage of cases can readily be ascribed to this cause; and it is evident that the Fraenkel-Weichselbaum pneumococcus is a constant accompaniment of genuine croupous lobar pneumonia in man, and, it may be added, according to the findings of many authors, that these bacteria are present in large quantities and, as a rule, unmixed with other micro-organisms."

The correctness of this fundamental statement cannot be doubted, notwithstanding that lately it has been shown that the vitality of Diplococcus pneumoniæ is not as feeble as was formerly supposed. Foà could preserve the organism fifty to sixty days, provided he obtained the blood from the infected rabbits as soon as the latter died or were about to die, still cultivated it for twenty-four hours in order to increase the diplococci, and then allowed it to remain in the dark under a cool temperature (65° C.—149° F.). Similarly, Emmerich has shown that pneumococci retained their power of development and virulence for months if kept in bouillon, provided they were cultivated for some days in the thermostat in one-half to one liter of bouillon and then kept at ordinary temperature in the dark, and then employed for the transmission of the precipitate. As a rule, this transmission from bouillon cultures one to two months old may even then be successful if the precipitates from two well-developed test-tube bouillon cultures are placed in a new bouillon solution (about 15 c.c.). He comes to the conclusion that among the pneumonia cocci there are also, if only very few, more permanent forms present. Fraenkel and Reichl, with the aid of their own methods, have succeeded in preserving the diplococci for a long time.

Although it must be admitted that the etiologic significance of Diplococcus pneumoniæ is strongly supported by the frequency and the relative regularity with which it is found in croupous pneumonia, the proof of a true specific action is still lacking. Diplococcus pneumoniæ does not necessarily cause characteristic croupous pneumonia when it becomes pathogenic in the human or animal body; it may also cause other pathologic conditions which have nothing in common with pneumonia, and for this reason a pathologic condition of organs seems to be necessary for Diplococcus pneumoniæ to develop its activity.

Diplococcus pneumoniæ is unquestionably present in meningitis which arises independent of pneumonia, in cerebrospinal meningitis, in endocarditis, in cases of primary pleurisy, peritonitis, endometritis, and purulent arthritis. Even Weichselbaum reports these findings. The latter, later on, was so extensively confirmed that there can be absolutely no doubt of it. I myself in the year 1880 demonstrated micrococci in the exudate of two cases of cerebrospinal meningitis, where the organisms were partially free in the pleura and partially within the pus-cells. Many of the individual cocci showed marked motility. I found similar forms in the blood, in the heart, in the splenic pulp, and in six areas in the lungs which were present in both cases and were hardly the size of a hazelnut and which had exactly the appearance of a lobar pneumonia in the stage of gray hepatization. I believe these areas were secondary. Later Leyden and Leichtenstern reported similar findings. The first observer is disposed to consider idiopathic cerebral and the cerebrospinal meningitis as exclusively due to the influence of Diplococcus pneumoniæ, and septic meningitis arising after erysipelas or trauma as due to Streptococcus. According to my own observations, I can thoroughly agree with the latter assumption. While, on the one hand, it can be considered as proved that Diplococcus pneumoniæ may produce a great variety of diseases in man; on the other hand, it has been established by experiments on animals that this micro-organism in at least a great many cases is hardly able to cause a pure pronounced pneumonia which is entirely identical with that of man and characterized by its exclusive involvement of the lungs. Only in a few cases Fraenkel as well as Weichselbaum succeeded in causing pneumonic processes by the intrapulmonary injection of the pneumonia culture. This cannot be accomplished with certainty, as Fraenkel himself admitted (compare Baumgarten, p. 251). Gamaleia, by pulmonary injections of the diplococcus, caused typical fibrinous pneumonia, it is true not in mice and rabbits, but in dogs and sheep. For the purpose of obtaining these positive results, cultures were injected which were increased in their virulence by successively passing them through rabbits. By the inhalation of moistened dust containing cocci Weichselbaum succeeded in bringing about a fatal infection in mice, but the lungs showed themselves intact. Subcutaneous injections proved entirely negative. Similar unfavorable results were obtained by experimenting with the sputum of pneumonia patients and with pieces of the lung taken from persons who had died from pneumonia. Kühn likewise did not obtain any positive results by the subcutaneous injections of sputum from patients afflicted with pneumonia. Griffini and Cambrini, experimenting with pneumonic sputa, only succeeded in producing death with the symptoms of septicemia. Mendelsohn did not obtain any positive results with inoculations of pneumonic sputum. The animals either remained alive or died without the development of the local pulmonary lesions. I have never succeeded in producing a croupous pneumonia in healthy rabbits by subcutaneous or intrapulmonary injection of pneumonic sputum. The principal results were only pleurisy and pericarditis. The rabbits that were inoculated subcutaneously or by intrapulmonary injection with pure cultures of pneumonia cocci remained alive. Only Salvioli states that he has produced croupous lobar pneumonia in guinea-pigs by tracheal injection of exudates from the lungs of those dying from pneumonia and those that contained encapsulated cocci.

According to my opinion, the almost constant failure to produce croupous pneumonia is explained by the fact that local conditions—that is, pathologic conditions—must be present to render possible the permanent deposition and multiplication of Diplococcus pneumoniæ. This seems to be sufficiently proved by a series of experiments which were reported as early as the year 1884. I believe I should repeat here what I reported at that time:

Experiment 1.—On February 10, 1884, I rubbed up a piece of a gray hepatized lung in water, and, having filtered the fluid through linen, injected it by means of a Pravaz syringe into a vigorous, pregnant rabbit. After two days the animal aborted, and in six days it was found dead. Postmortem examination showed a purulent infiltration of the tissue in the region of the point of injection, suppurative pericarditis, and peritonitis with slight fibrinous adhesions between the intestines; the spleen moderately enlarged; the uterus externally intensely red, almost bluish-red, and its mucous membrane markedly reddened; around the same there were six circumscribed areas of discolored purulent serum the size of a ten-pfennig piece. The pus in the pericardium and peritoneum, as well as in the placental areas of the uterus, showed a great number of micrococci, which resembled in every respect pneumonia cocci.

Experiment 2.—I made the same experiment on February 21st with a piece of a gray hepatized lung that I had prepared in the same way. On the 23d the animal gave birth to three dead fetuses almost at full term. The animal died on the 24th. The postmortem examination was conducted two hours after death. At the point of injection there was purulent edema. With the exception of the uterus the internal organs showed no abnormality. In the uterus I found still another fetus whose placenta was loosened in part from its point of attachment. The uterus and placenta showed a whitish discoloration. Of the three remaining placental areas, which occupied the right half of the uterus, two had a discolored hemorrhagic appearance and the third a well-pronounced diphtheritic appearance. The grayish-white masses which were here found could be partially scraped off. All the mucous membrane of the uterus between these placental areas was markedly swollen, and especially in comparison with the left half of the organ; the latter had also a hazy appearance and was covered with numerous punctate hemorrhages.

Microscopic preparations were made from the areas surrounding the point of injection in the diphtheritic placental area and from the diseased uterine mucous membrane; identical micrococci were generally found arranged in pairs. Occasionally three to four diplococci formed a chain, the whole having a pale envelope.

The result of these experiments was, as a matter of fact, almost strikingly as I had expected. In one rabbit stall in which in the ten years of its existence no rabbit had ever died in consequence of giving birth to a litter, there died after the injection of a fluid derived from a pneumonic lung, two pregnant animals shortly after aborting. The one which had lived somewhat longer showed upon macroscopic examination a diphtheritic inflammation of the placental areas, a peritonitis and pericarditis; the other showed an exquisite hemorrhagic endometritis and also diphtheria of the placental areas. The pathogenic micro-organisms found in the diseased areas were identical with one another as well as with those which were employed for the injection, and, what is still more significant, completely resembled those which are found in human diphtheritic puerperal endometritis. The identity of the micrococci obtained from a fibrin-clot which has been expectorated by a pneumonia patient, with micrococci from the endometrium of a rabbit that had died of diphtheritic endometritis, and with micrococci from the endometrium of a woman who had died of diphtheritic endometritis, was demonstrated under the microscope with fuchsin as a stain. I do not believe there is any doubt about the microscopic identity of these three sets of micro-organisms.

Experiment 3.—Next I wanted to be satisfied that the change taking place in the pneumonic lung in the dead body did not materially influence the experimental result. I therefore employed the sputa of a patient afflicted with pneumonia, for in this expectoration the diplococci can as well be found as in the lungs of cadavers. As it incidentally proved quite convenient, I injected one cubic centimeter of pneumonic sputum into a rabbit two hours after the animal had given birth to six young ones. Forty-two hours thereafter the animal died. At the point of injection there was a purulent infiltration the size of a dollar, and close thereby the connective tissue was swollen and appeared gelatinous. The glandular substance of the mammæ contained considerable milk, the latter being evacuated in great quantity upon incision. Lungs and heart showed no abnormality. The spleen was not enlarged. The liver showed in its dark-brown substance several punctate grayish areas. Punctiform hemorrhages were found in the kidneys. The vagina was pale; the mucous membrane of the uterus was swollen throughout, dark red, and covered by numerous hemorrhages the size of a pin-head. These changes were most marked at the placental area, which also contained a few small blotches of a discolored purulent appearance.

The dry preparations of subcutaneous connective tissue stained with fuchsin, and preparations of the blood, of the liver, and of the inner surface of the uterus, showed numerous micro-organisms, almost exclusively in the form of diplococci. Most of the latter possessed a light pale envelope.

Experiment 4.—A piece of liver taken from the just-described rabbit which had died of puerperal endometritis in consequence of subcutaneous injections with pneumonic sputa, was rubbed up in distilled water, filtered through linen, and subcutaneously injected by means of a Pravaz syringe into a moderately large pregnant rabbit. After thirty-six hours four young rabbits almost at full term were found. The animal died in five days. The areas surrounding the point of injection and a portion of the mammary tissue on the right side were infiltrated with pus. A large quantity of a serous fluid was found in the pericardium. The pericardium itself appeared slightly turbid; a moderate quantity of serous fluid was also found in both pleural cavities. The lungs could contain air without difficulty.

The intestines were adherent by abundant but easily detachable fibrinous masses; the uterus was irregularly colored a markedly livid red, and a portion of the same, where the wall was thicker, showed a discolored grayish-white appearing serosa. Corresponding to this area, the uterus contained a remaining placenta whose uterine surface of attachment appeared discolored and gray. The placental tissue itself had up to a certain depth a similar appearance, which could not be removed by scraping away the upper layers. The remaining three placental areas had a discolored hemorrhagic appearance. Macroscopically the liver and the spleen appeared unchanged; the kidneys were large, and in some places covered with red spots.

Dry stained preparations from the surface of attachment of the remaining placenta, from the placental areas of the uterus, as well as from the blood and the liver, showed very numerous diplococci.

This experiment may, then, be considered as representing a pure culture of bacteria in the animal. The fluid obtained by rubbing up a piece of the liver from the rabbit that had died of diphtheritic endometritis, and which contained the specific diplococci, sufficed to cause the same disease in another pregnant rabbit, with a marked increase in the number of the micro-organisms.

It may, therefore, be considered as a fact that the subcutaneous injection of pneumonia cocci causes a diphtheritic endometritis independent of the fact whether these cocci are found in the dead pneumonic lung or in the sputum of pneumonia patients, or in the pure culture of the blood of an animal that has died of puerperal endometritis caused by such sputum.

It therefore was possible for me by these experiments to cause such grave pathologic changes as to produce the death of the animals experimented upon; but the lungs were not affected—only such an organ in the animals which at the time of infection with the pneumonia cocci offered the greatest disposition for the adhesion of the organism, namely, the uterus. In three cases the animals were experimented upon shortly before they gave birth to a litter, and one had had young ones two hours previously. In all cases a diphtheritic endometritis resulted. That the diplococcus of pneumonia was the cause of this affection can be deduced with certainty from the fourth experiment; for while in the first two experiments pieces of pneumonic lung taken from the cadaver were employed, and in the third experiment the sputum of a pneumonia patient, the fourth experiment was performed with a portion of liver the blood of which on microscopic examination was found to contain only a pure culture of the diplococcus.

As a confirmation of this experimental result, I can add from the literature an observation on man that was made by Czemetschka. This author describes a patient in whom, during pregnancy, an infection had taken place, and, as postmortem examination showed, from the nasal cavity. Not only a pneumonia developed, and later on meningitis and endocarditis, but the fetus also was infected with pneumonia by the mother, and, in consequence of this acquired pneumonia, died soon after birth. In the patient herself the infection had been carried by the blood stream from the placental site, and had given rise to a suppurative metro-lymphangitis and a purulent infiltration of a vaginal laceration. One and the same cause, the Diplococcus pneumoniæ, had thus caused the various pathologic conditions in the organism of the puerperal woman. I myself have, on the strength of the experience gained from these experiments, immediately instituted intra-uterine treatment (carbolic acid irrigation), and have seen recovery follow in two women who had suffered from pneumonia while far advanced in pregnancy, and who subsequently gave birth to children.

Having therefore established the fact that Diplococcus pneumoniæ is never absent in croupous pneumonia, and that, according to our present pathogenetic views, the diplococcus can be considered as the cause of a particular form of pulmonary inflammation, —namely, the croupous,—notwithstanding that experiments upon animals do not yet fully substantiate this, and that accessory powers are necessary to permit the virulence of the organism to develop locally; the question arises, By what path does it gain access to the body? That the virus in localized diseases of the respiratory organs—therefore, also, that of croupous pneumonia—is inhaled, has been already declared by Cohnheim (p. 224). Baumgarten states that it can hardly be doubted that under natural conditions the pneumonia virus enters the lung from the respiratory passages, remains there, and only later becomes localized in other organs. At least there is an absolute lack of proof that in genuine croupous pneumonia of man the pneumonia virus is primarily developed in the blood, and from there attacks individual organs, especially the lungs.[252] Ribbert also assumes that the pneumonic process has its inception in the respiratory passages. The cocci are found chiefly in the interior of the cells. This author, it is true, bases his view on the assumption that the pneumonia begins in the bronchioles and alveolar passages, and from them spreads to the surrounding regions.

In support of these views is the finding of the diplococci in the pneumonic exudate. Orthenberger, with Weigert's method of staining, has shown that in a series of fatal cases of croupous pneumonia taken at random and caused by the diplococcus or streptococcus of pneumonia, the cocci were situated in the cellular alveolar exudate and within the cells themselves; as well as that in these cases, even if no complications in other organs had occurred, the micro-organisms were to be found with certainty in the white blood-corpuscles and also in the vessels of the body. Conrad Zenker found numerous pneumonia diplococci in the leucocytes in the alveoli as well as in the interstitial tissue in pneumonia with abscess formation.

It is easy to determine whence the diplococci come that enter the lung from the respiratory tract—namely, from the respiratory tract itself, for they are so frequently found in the mouth, in the nose, and in the air-passages of healthy individuals that it can confidently be asserted that they are only waiting for a chance to become pathogenic in the lung.

Netter demonstrated Diplococcus pneumoniæ in the saliva. In a very extensive series of experiments v. Besser, by examining 81 times the nasal mucous membrane of 57 men, found Diplococcus pneumoniæ 14 times, and proved it to be such by pure culture as well as by transmission into animals. In ten cases shortly after death the bronchial secretion was examined under all precautions and Diplococcus pneumoniæ found three times. The cases were: A fracture of the cervical vertebra, tuberculosis of the peritoneum, and a case of enteric fever. Bein considers that the number of people in whose mouths the pneumococci are present is 30%. The reports of individual investigators vary between 15% and 30%. Marfan states that in all cases of bronchitis he has found the pneumococci in the secretions. According to Polynère, pneumococci are found even in normal lungs. [In September, 1880, Sternberg isolated the diplococcus from his own saliva and inoculated rabbits; Pasteur discovered the same organism in December, 1880.—Ed.]

In consideration of these results, it must be admitted as not proved, that the pneumococcus, when it does cause pneumonia, first circulates in the blood and then enters the lung, and the possibility of such a phenomenon must be thought applicable only to rare cases. But, on the other hand, it must be considered as undoubtedly correct that the cause of the disease enters the alveoli through the air-passages. The further distribution of the pneumonia cocci from the diseased lung very likely takes place principally by means of the circulation. Although Weichselbaum considers as the cause of meningitis the direct continuation of the process through the cellular tissue of the neck to the meninges, nevertheless, considering the localization in organs that are further removed,—for instance, the kidney,—it is to be supposed that dissemination by the blood stream is far more frequent. It follows, from the results of examining the blood or cultures from the blood of pneumonia patients, that in the course of every pneumonia varying numbers of pneumonia diplococci are present in the blood for a greater or lesser period. Weichselbaum had already shown the pneumococci in the blood of patients ill of pneumonia. Belfanti examined the blood bacteriologically in numerous pneumonia cases, and six times during life could verify in these the presence of Fraenkel's diplococcus. For purposes of examination he usually took 30 to 50 c.c. of blood from the veins. Only in one case was it possible to find pneumococci microscopically in blood taken from the finger, and then subsequently to cultivate the same. Casati found in pneumonic individuals, both in the blood taken from the heart cavities after death and in the circulating blood during life, Fraenkel's diplococci constantly. During life they were present in the blood from the second day of the disease, both in the cases which ended fatally and in those that recovered. Righi, like Vincenzi and Quadu, not only found the diplococci in the blood, as did the two last-named authors, but also found them in the urine and feces. From this he deduces the fact that the excreta are also means of transmitting the disease.

The results of bacteriologic investigation up to the present time may be summed up as follows:

The cause of croupous pneumonia is the diplococcus discovered by Fraenkel and Weichselbaum. Under normal conditions its habitat is in the upper respiratory passages. But as it is not the cause of pulmonary inflammation alone, but can also give rise to other diseases, and as it commonly causes in animals inflammatory and, more correctly speaking, septic diseases, provided it does not attack organs with a local disposition,—as, for instance, the puerperal uterus, where it causes a true puerperal endometritis,—it therefore requires for its growth and for the development of its pathogenic influence in the lung certain auxiliary causal factors which will be discussed in the following section.

[It is best that the author's exposition of the subject should stand, although it would seem that a clearer conception of pneumonia would be had if the lung process were considered to be one of many manifestations of a *pneumococcus infection.* In this manner we believe the clinical course, the complications, and the sequels would be clearly understood. A clinical picture that failed to correspond with that described as pneumonia, would thus be better appreciated. There is some reason for the existence of the terms "lung fever" or "pneumonic fever" under these circumstances. Moreover, the fact that with improvement in our technique we are able to recover from the blood of patients with pneumonia the diplococcus of Fraenkel in an increasing number of cases, suggests the possibility of the invasion of that tissue in every instance.

The diplococcus of Fraenkel has been found in varying numbers in the blood of pneumonia patients by numerous investigators. The percentage of cases in which the organism was found varies within wide limits, from 10% to 50%; the highest proportion is reported by Silvestrini and Sertoli, who found the diplococcus in 15 out of 16 cases.

A similar diversity obtains in the opinions as to the prognostic significance of the presence of the diplococcus in the blood; but, on the whole, it appears that the prognosis is grave if large numbers are found. The discovery of isolated diplococci in the blood is of no prognostic importance.

Beco reports one case of pneumonia due to Friedländer's bacillus in which there was a general bacillemia.

Baduel arrives at the following conclusions:

The diplococcus of Fraenkel is present in the majority of cases.

In many cases it is not pathogenic for animals.

The number of the organisms bears a relation to the gravity of the local process.

Virulence is usually associated with acute lesions.

The persistence of the diplococcus in the blood is a common phenomenon and not a complication to be dreaded.

The diplococcus may localize itself in the blood primarily or secondarily; in the latter case the diplococcemia is much more grave.

(Authorities: Kohn, Pani, Pieracconi, Berghius, Silvestrini, Sertoli, Baduel, Beco.)—Ed.]

AUXILIARY CAUSES OF PULMONARY INFLAMMATION.

The disposition to acquire croupous pneumonia exists over the whole surface of the earth; nowhere do local conditions prevent the occurrence of the disease. This can be deduced with certainty from the numerous figures of Hirsch. Neither has climate any particular influence upon the frequency of the occurrence. "For in the most northern regions of Europe inflammations of the lung are of but moderate frequency, at least not more frequent than in many other more southerly situated regions in the same portions of the earth; while, conversely, there are reports of the frequent occurrence of the disease from southern countries" (Hirsch, p. 79).

The view of v. Jürgensen, that the geographic distribution of pneumonia is entirely different from that of catarrh and bronchitis, appears to me untenable. He states: "The latter increase in frequency the more one ascends from the tropics to the higher latitudes; this is not the case in pneumonia. Just as little, however, is the reverse true, as has been contended from many sides" (p. 13). Against this view the facts established by Hirsch can be adduced. He states: "In Italy catarrhal diseases of the respiratory organs, as Parola expressly declares, belong to the *malattie communissime,* and are mostly found along the coast as well as on the plains of the interior and in the mountain districts" (p. 2); and in another place (p. 79) he states that numerous earlier observers have called attention to the frequency of pneumonia in many regions of Italy, and that these statements are supported by the high mortality of "inflammation of the lungs" in the middle and southerly districts of Italy, reported by Sormanni, who says: "If one considers the winter climate, particularly of middle and lower Italy, one would think that diseases of the chest would be unknown, or at least rare in those regions. Instead of this, it appears from the public statistics of the civilian population as well as of the militia that these diseases arise frequently enough."

While it is evident from these numerous statistics that climate is a minor factor in the incidence of pneumonia, it appears that racial or national conditions have a marked influence upon the frequency of the disease. Individuals migrating from northern latitudes to subtropic or equatorial regions seem to enjoy a relative immunity from inflammations of the lungs; on the other hand, the inhabitants of the tropics and, above all, the negroes are particularly subject to the disease not only after locating in colder regions, but also in their native country. How far this fact may be explained by a congenital or acquired predisposition in the colored races, and especially in the negro race, and how far the wretched hygienic conditions in which they live, or the notorious indifference with which they expose themselves to the elements, may be determining factors, cannot be positively decided (Hirsch, vol. III, p. 103).

On an average, pneumonia amounts to 3% of all diseases of man. Of internal diseases alone, pneumonia lays claim to 6.4% in Germany, England, and France (v. Jürgensen, p. 12). Nevertheless the latter figure appears to be estimated somewhat high.

As we do not yet possess morbidity statistics of the total population, we must confine ourselves to the reports from hospitals and public clinics. Von Ziemssen estimates the percentage of cases at 3%. In the hospital at Altstadt, from January 1, 1880, to April 1, 1896, I had, among 36,540 patients admitted, 1501 pneumonia cases; this comprises 4.1% of all internal diseases. [In the United States in 1890 the death-rate per 100,000 population was 186.94. There has been a marked increase in frequency during the past twenty years. Reynolds believes this is due to the prevalence of influenza. Osler states pneumonia has increased in frequency in Baltimore; and Folsom shows a like increase for Massachusetts, and Norris claims an increase in this city.—Ed.]

Especially noteworthy and important for the comprehension of the predisposing causes is the fact that the number of pneumonia cases bears a direct relation to seasons, and to the prevailing condition of the weather. Empiric as the statement may sound, it is nevertheless true that the prevalence of pneumonia is directly dependent on the prevalence of unequal and changeable meteorologic conditions, especially on the occurrence of abrupt and marked changes of temperature at a time when the mean temperature is very low. This coincides entirely with the distribution of my cases of pneumonia as regards the individual months. In 1501 cases of pneumonia, the distribution was as follows:

January	174	April	189
February	164	May	179
March	206	June	94
First Quarter	544 = 36.2%	Second Quarter	462 = 30.7%
July	62	October	96
August	49	November	103
September	71	December	114
Third Quarter	182 = 12.1%	Fourth Quarter	313 = 20.8%

Therefore in the most favorable season, the third quarter (July, August, and September), only one-third as many cases occur as during the most unfavorable season (January, February, and March). [Of 195 cases under the care of my colleagues and myself, 69 occurred in the first quarter; 57 in the second quarter, 31 of which were in April; 18 in the third quarter; and 51 in the fourth quarter.—Ed.]

It would naturally be of great value to analyze the empiric conception in reference to the increase of pneumonia during the unfavorable season, and to determine, as it were, the component parts. But, in spite of repeated researches in this direction, it has not been possible to determine them with certainty.

The most important factor, in all probability, is a rapid change of temperature. Huss says: "During the spring months the most rapid changes of temperature are noted, not only from one day to the next, but during the different hours of the same day, and especially between day and night. These changes appear to be the plainest predisposing causes of the development of inflammations of the lungs, and as they are more conspicuous in some years than in others, this probably explains the fact that in some years there are more cases of pneumonia than in others."

A further proof of the correctness of the supposition that marked changes in the temperature are of particular importance in the development of the disease is found in the fact dwelt upon by Hirsch that the maximum incidence bears a certain relation to the temperature changes arising at particular seasons in certain localities. In the countries situated in the higher latitudes (Russia, Sweden, Denmark, Germany, England, the northern districts of France, the northern United States of America), in which the most abrupt changes in temperature occur in spring, the maximum of the frequency of disease is noted in the spring. On the other hand, in the warmer and subtropic countries (Italy, the islands of the Mediterranean, Greece, Algiers, the southern United States of America, Chili, Peru), in which the previously mentioned meteorologic influence makes itself especially felt in the winter, the latter represents the especial pneumonia season (vol. III, p. 95).

The influence of changes in temperature, which, upon the basis of these reports, have been looked upon as etiologically important conditions, has also been proved experimentally. Lipari has given the proof that animals which, after an injection into the trachea of pneumonic sputum have remained well, were affected by pneumonia if they were subjected to the influence of cold before these injections were made. Of eight animals which were experimented upon in this manner, six died showing decided pneumonic infiltration. He presumes that cold paralyzes the epithelial cells of the bronchial tubes and produces a swelling in the mucous membrane—two pathologic processes which, in his opinion, favor the entrance of the infectious principles into the alveoli.

Further, two observations of Bein are worthy of being mentioned at this place. He was able to demonstrate both the indirect cause—a plunge in cold water in one case, and ammonia poisoning in the other—and the direct cause: namely, the presence of virulent pneumonia bacteria in the oral mucus, in one case before the onset, and in the other at the very beginning of the attack.

Accordingly it is a fact worth considering that after a thorough wetting, as is often said, pneumonia not rarely follows. I have noted several such cases, which curiously enough occurred in the favorable season. This special injury at any season offsets the unfavorable influences of the weather during the winter, through which a large number of persons are predisposed to pneumonia, and is of more importance than the influence of an excessive rainfall.

Living in localities exposed to severe winds or to great drafts is not without influence upon the production of the disease. Welch mentions the following fact: "Pneumonia occurred in a battalion which had been stationed for six years at a Mediterranean port, and was transferred from there to North America during raw winter weather. Of these troops, 330 men were stationed in a frame building, which no doubt would have been very habitable in the summer season, but in the winter did not give sufficient protection against the weather, as it was exposed on every side, and the thin walls through their numerous cracks gave the wind free access to the interior. Two hundred and fifty-six men were stationed in well-protected barracks and 66 married soldiers found quarters in the city. Among the men stationed in the wooden building 38 developed pneumonia, whereas among the other soldiers only three had pneumonia, which might easily be referred to the influence of the weather. The severest cases among those in the wooden building occurred in those living in the upper rooms, which were most exposed to the sharp winds coming from the north and west. The disease unquestionably arose from the fact that during the night the body was exposed to a draft and to the general low temperature."

These facts that were noted among a large number of troops no doubt occur more frequently in individual cases than is generally reported. I only find Finkler describing one case which might be counted in this category. A robust man who in an overheated room was perspiring freely was exposed to a cold blast by the door being suddenly blown open when the temperature was at the freezing-point. He was at once attacked by dyspnea, and after a few hours a severe chill occurred, which was followed by pneumonia.

The direction of the wind is without importance. In Finkler's collection Grisolle noted the most pneumonias occurring during a northeast wind; Schramm during an east wind; Sturges during a north and northwest wind.

Among the laity the view is prevalent that small children should not be carried in the open air during the prevalence of easterly winds, as under such circumstances they might easily contract inflammation of the lungs. I have not regarded this rule, and have found no evil results to follow; only if dreary, foggy, cold, wet weather was prevalent have I not permitted children to go out into the open air.

The influence of the barometer may also be regarded as unproved. According to v. Jürgensen, a high barometer coincides with a lessened frequency of the disease. He believes that he has noted in Kiel that pneumonia becomes more frequent if the low barometric pressure, which at that place is fairly constant during the prevalence of northeast and east winds, is suddenly interrupted.*

All faith in the influence of the barometer must be lost when we hear diametrically opposed opinions in reference to the prevalence of the disease with respect to the state of the barometer based on practically the same clinical material. Knövenagel says: "One of the principal points in the explanation of the frequently arising and almost epidemically arising appearance of inflammation of the lungs is not a sudden, sharp change, not severe wind, not a thorough wetting, but dry air continued for a long time with high atmospheric pressure, which from day to day and from week to week is inhaled, and favors the introduction of miasmatic poisons, so that eventually, when one has been exposed to such poisons in sufficient number, in the one case sooner, in the other later, the infection goes so far that the typical chill arises."

Apart from this peculiar conception that the atmospheric pressure upon the respiratory surface favors the further introduction of miasmatic poisons, Riebe, upon the basis of analogous investigations, arrives at a diametrically opposite result. In the winter of 1879–80, which for Knövenagel in Cologne and for Riebe in Posen was characterized by high atmospheric pressure, only 27 cases of pneumonia occurred in Posen. On the other hand, during the winter of 1878–79, when the barometer was much lower, 50 cases occurred, and in the winter of 1880–81, 60 cases were noted. "It seems that an auxiliary cause of the disease here in Posen is found in excessive humidity" (Riebe, p. 323). Brunner declares as a result of extensive investigations: "It is shown that the three months in which the most cases arise are characterized by a minimum of atmospheric pressure and very low relative humidity."

The conditions of the weather which have here been noted, of themselves have, however, an increased disastrous influence upon the production of pneumonia in the human organism only when the body finds itself under abnormal conditions. Especially the individual disposition as regards pneumonia must be placed in the foreground more than in any other infectious disease. The duration of such a disposition may be very brief and transitory. One of the chief predisposing factors is either unaccustomed or excessive muscular exertion, which may give rise to an enfeeblement of the entire muscular system, and, in connection with this, to an increased function of the sweat-glands.

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* See also "Etiology of Pneumonia," Baker, "Reports Mich. State Board of Health."

This is shown by the excessively high incidence of pneumonia among recruits. Gründler declares that the origin of the numerous pneumonic affections which he has noted in the garrison of Magdeburg from the first of October, 1873, to the end of June, 1874, was due less to the influences of the weather than to the arrival and education of recruits. Whereas only two or three cases occurred in October and November, after the third of December, at which time the recruits arrived, the cases suddenly became more numerous, and during December and January 14 were noted in each month. During February and March the same number occurred, gradually becoming less in April and May, until in the month of June the normal number, four, was reached. The complete strength of the garrison was 6008 men, consisting of—

Recruits or men in the first year of service	2226;	of whom 48 men	= 2.15% were attacked.
Soldiers, second year of service	2076;	" " 17 "	= 0.81% " "
" third " " "	1031;	" " 7 "	= 0.67% " "
" after the third year of service	675;	" " 1 man	= 0.14% was "

Among recruits, therefore, there was three times as great a chance to acquire inflammation of the lung as among men in the second and third years of service.

In an analogous observation Knövenagel arrives at conclusions which are far removed from the actual data at his disposal. He believes that the constant pressure of the atmosphere on the lungs offers the best explanation for the great frequency, not to say epidemicity, of inflammations of the lungs. No sudden sharp change, no severe winds, no wetting, but only a high atmospheric pressure during the time of long-continued dryness of the air, which hypothetic results have already been mentioned, are responsible; upon this basis, and perhaps also quickened through coincident causes,—for example, a thorough wetting, upon which, and especially by the patient, all the fault is laid,—the disease begins and reaches its full development rapidly.

The most careful work upon the question, so important, in my opinion, what causes are responsible for the undoubted frequency of the disease among recruits, we owe to Riebe. The total number of cases treated by him during the years from 1873 to 1881 show the following percentages for the individual months:

November	3%	May	11%
December	7%	June	7%
January	11%	July	4%
February	13%	August	3%
March	19%	September	2%
April	14%	October	1%

The percentage of recruits affected in comparison to the total number of cases of pneumonia, according to the separate months, is as follows:

November	75%	May	52%
December	60%	June	50%
January	54%	July	63%
February	59%	August	57%
March	60%	September	47%
April	46%	October	40%

From these figures the significant fact is to be deduced that the sick-rate among the recruits as compared with the general sick-rate presents marked variations during the individual months. Unless it is conceded that individual overexertion is a potent predisposing factor, it is difficult to explain why the proportion of recruits to seasoned soldiers should not at least be the same at every season of the year. Instead, however, it appears that in the month of November, when the total sick-rate is extremely low (3%), the recruits furnished 75% of the cases, the highest contingent of the entire year. Any one who is not absolutely and wilfully blind cannot fail to see that the explanation of this state of affairs is to be found in the excessive demand on the powers of the young recruit and in the unwonted bodily exertion incident to the first year of service.

Riebe correctly deduces from his observations that the occurrence of croupous pneumonia in Posen was due neither to quarters, their surroundings, temperature, pressure, nor humidity of the air. On the contrary, he declares positively, first, that the disease occurs preferably during a certain season, and that, in spite of the various geographic situations of the observed persons, the disease occurs with great regularity during the spring; second, that preferably recruits—that is, persons who have not yet become accustomed to the climate and who are not yet used to military exertions—contract the disease (p. 334). On the other hand, it is not to be forgotten that the younger persons take part in the drills in disproportionately greater numbers; naturally, they take part in all exercises, while of the older men the hautboy players, mechanics, orderlies, and soldiers on special details are usually excused.

Witte arrives at similar numerical results. He reports, concerning 523 cases of pneumonia which occurred among soldiers from 1881 to 1886, that the maximum occurred during the months of February, March, and May; in November 74% of the recruits were affected, and in December 80%. The total number of recruits equaled only a little more than one-third of the total number of men.

Additional proof of the injurious influence of bad weather combined with excessive bodily exertion is furnished by the following facts and statistics. During the epidemic in Magdeburg, described by Gründler, not a single case of pneumonia occurred in the field artillery, "and the men in that branch of the service were not forced to drill on foot as much as other troops." In Posen the sick-rate was at least somewhat lower in the field artillery (Riebe). In Wesel during the eight years the battalions of infantry had between 32 and 47 cases, while in the two divisions of artillery the numbers were only 18 and 19 respectively (Knövenagel). At the Third Congress for

Clinical Medicine, Fräntzel stated emphatically that, after analyzing the material at his disposal in the capacity of military physician, he was forced to the conclusion that the highest incidence of pneumonia coincides with periods when the men spend much of their time in the open air and are exposed continuously to the bad influences of the weather.

Similar exact reports have not been made among civilians. Individual observations concerning the influence of uncommon exertions undoubtedly have been made by many physicians, but they have not become common knowledge. Riesell, a careful investigator of the etiology of croupous pneumonia, quotes himself as an example. One day he was obliged to traverse a precipitous road in visiting a patient suffering from pneumonia, and, being particularly busy, hurried, and, without waiting to allow his heart to recover from the unwonted exertion, made a thorough examination of his patient. On the evening of the same day he felt much depressed and on the third day thereafter contracted a double pneumonia. I myself have seen a fourteen-year-old boy contract a grave pulmonary inflammation twenty-four hours after he had run one kilometer with a horse-car on a wager, having run as fast as the conveyance. I also include among these cases that of Brunner, although he himself considers it as belonging to those occurring epidemically. The seventy-two-year-old husband of a woman sixty-four years of age suffering from inflammation of the lungs, and who occupied the same bed-room, nursing his wife, "and who frequently had to run three miles [English] to call the physician," likewise contracted pneumonia three days after one of these runs.

The statement of v. Jürgensen, that people living in the open suffer less from pneumonia than those confined indoors, would prove exactly the opposite of what has been observed in the army if the foundation for this were not very weak and were not in utter contradiction to the experiences of Grissolle. The latter states that men working indoors contract pneumonia 2½ times less than those who are constantly employed in the open air. Likewise women remaining in the open air contract the disease more than other women.

If, however, the great pneumonia mortality in prisons is to be taken as a proof for the frequent affliction of people who are confined indoors, then the error lies in the consideration of forms of pneumonia which have absolutely nothing in common with genuine croupous pneumonia. Most of the pneumonias occurring in prisons are of the so-called infectious variety, which are to be classified among the atypical pneumonias, and which will be considered by themselves.

I have attempted to solve this question concerning the prevalence of pneumonia among civilians by compiling in a statistical manner the total number of cases treated at the Magdeburger Alstädter Hospital from 1892 to 1895.

Of those admitted during the years 1892 to 1895 inclusive, the following contracted pneumonia:

THOSE ENGAGED IN INDOOR OCCUPATIONS.

102	officials	4
105	bakers	4
22	brewers	2
9	glaziers	2
7	goldsmiths	3
199	merchants	10
66	waiters	1
29	metal-workers	2
18	harness-makers	4
126	tailors	7
116	shoemakers	8
138	joiners and coopers	3

Therefore out of a total of 930 men in the above class, 50 contracted pneumonia, a percentage of 5.3%.

THOSE ACTIVELY ENGAGED IN THE OPEN AND AT HARDER WORK.

1080	laborers	87
83	butchers	7
36	gardeners	3
41	coachmen	7
59	masons	2
54	sailors	1
168	locksmiths and blacksmiths	16

Among the 1521 men of the above class, 123 contracted the disease, a percentage of 8%.

[*Occupations.*—Of 500 cases Norris determined the following:

MALES.

Indoor occupations			Mortality	26.0%
Outdoor “			“	29.0%
Laborers	122;	died, 42	“	34.0%
Teamsters	8;	“ 4	“	50.0%
Stevedores	13;	“ 6	“	46.0%
School-children	61;	“ 5	“	8.2%
Children under 3 years	11;	“ 1	“	9.0%

—ED.]

From these data one fact at least may be deduced with certainty: the relatively more frequent affliction of those whose vocations exposed them to the elements. In spite of the small numbers, the irregular proportion between coachmen and butchers, in contradistinction to tailors and shoemakers, is conspicuous. Among the coachmen that were admitted, pneumonia occurred in one out of six, while among the tailors the proportion was one in eighteen. I will still have to leave undecided whether in the latter a Sunday dance, with the necessarily accompanying excesses *in Baccho et Venere*, did not have more influence in giving rise to the disease than the labor of the whole week.

While the individuals may contract a predisposition as the result of the pernicious influence of the elements, and while the facts alluded to have not yet been substantiated generally, another predisposing cause has been recognized during the last few years, particularly due to the careful investigations of Litten—namely, the influence of trauma upon the occurrence of pulmonary inflammation.

Andral has called attention to the probability of this causative relation. Grisolle has seen three cases of pneumonia occur after contusions. In one instance the sputa were purely bloody for three days.

Litten explains in a most detailed manner that lobar, croupous (or fibrinous) pneumonia may arise in consequence of a contusion of the thorax, without the thoracic wall itself showing the slightest lesion. He distinguishes these "contusion pneumonias" from traumatic pneumonia occurring in consequence of a stab or gunshot injury. The first variety is caused by a violent concussion which affects the lung and depends probably upon physical phenomena that are combined with the concussion of the organ. Among 320 cases he could trace the cause to a contusion 14 times (4.4%). Ordinarily these pneumonias develop one or two days after the accident. If in consequence of the concussion hemorrhages have taken place in the lung, lobar pneumonia is then superadded, the hemoptysis generally changes to rusty sputum.

Individual predisposition in contusion of the lung shows itself in the fact that areas are affected in which old pathologic conditions are present (adhesions, bronchiectases, tuberculous infiltrations, emphysema), quite independently of the area affected by the trauma.

Stern has made an exhaustive historical review as well as a complete statistical account of traumatic pneumonias. According to his compilation, the percentage of traumatic pneumonias varies between 4.4% (Litten found it 14 times among 320 cases) and 0.13%. (v. Jürgensen once among 768 cases.) I myself have not observed traumatic pneumonias any more frequently than v. Jürgensen; perhaps the number would have been larger if each case had been questioned as to a previous contusion. Few patients would volunteer the information, principally because the gravity of the clinical symptoms at the time hardly allow them to remember an accident that apparently had no direct consequences. I will call attention to one case of contusion pneumonia, particularly because it was carried into court to decide the question of accident insurance. Up to that time he had been a healthy man, thirty-eight years of age, a mason by trade. While pushing a wheelbarrow containing stones, he was injured by the left handle of the wheelbarrow striking him on the left side of the chest. On the evening of the same day, about ten hours thereafter, a pneumonia occurred, but upon the right side, the one opposite to the injured one. Seventy-two hours after the accident he died. Postmortem examination showed a pneumonia of the right base. Based upon the above-named experiences of Litten, that the pneumonia in no way need represent the side that is injured; and, furthermore, considering that the man was entirely healthy up to the time of the injury; and, finally, and principally because an eye-witness testified as to certain important symptoms which I considered could only be due to concussion of the lung—without external injury or internal laceration of tissue—and which could be observed, but could not possibly be invented, by a layman, I testified that the death of the man was due to an inflammation of the lung caused by the accident. [We have seen one case in which trauma and fright played equal parts, as the man was injured by accident due to a runaway horse. In the carriage with him were members of his family, hence the shock.—Ed.]

The symptoms observed immediately after the accident, which have so far never been mentioned, and which perhaps may be elicited in the future by taking a careful history, I now give, in the words of the eye-witness: "The mason D., as I observed, could probably, in consequence of the blow, get no air, pressed his hand against the right side of his chest, and opened the mouth for air. When he recovered, which occurred soon, he wheeled the barrow back to its place and continued his mason's work for about two hours longer. After his midday meal he wanted to resume work, but felt too unwell and had to relinquish it." [Harris * reported a case of chest injury followed immediately by clinical signs of pneumonia, finally ending as progressive tuberculosis in twelve weeks.—Ed.]

The association of Diplococcus pneumoniæ with the inflammations caused by trauma, and for which this organism has the same etiologic significance as in the non-traumatic pneumonias, has already been demonstrated by Weichselbaum. In two cases of contusion pneumonia he could show the diplococci.

Psychic conditions of depression, such as anger, grief, etc., are not without influence upon the occurrence of the disease. Riesell gives instructive and convincing examples of the occurrence of pneumonia after violent psychic disturbances. I myself have observed two cases in private practice in which violent anger and worry over financial losses gave rise to the disease. In both of these cases it was remarkable that the brain was affected to a marked degree. The most violent unrest existed, which increased gradually to furious delirium. [Two of our cases were in persons who had been robbed. Both terminated fatally and, as the author cited above observed in his case, with great toxic cerebral symptoms.—Ed.]

No influence upon the occurrence of croupous pneumonia can be ascribed to the inhalation of noxious gases. In such cases there is generally a deglutition pneumonia, as Nauwerck has shown, as in the case of the pneumonias following etherization. According to my observation, in the majority of these cases after the inhalation of the gases there occurred a condition of more or less lasting unconsciousness, during which the fluid in the mouth gained access to the respiratory passages.

On the other hand, the importance of some acute diseases in giving rise to a complicating pneumonia is well recognized. To this class of diseases belong especially enteric fever, typhus fever, puerperal fever, and intermittent fever. In the first-mentioned disease the occurrence of pulmonary inflammations is certainly caused by the typhoid bacillus itself, as was shown by Polynère, who demonstrated them in the pneumonic areas. But these changes have nothing in common with the croupous pneumonia occurring during the course of typhoid fever or at the beginning of convalescence. "The splenization and bronchopneumonic areas seen in the pneumonias produced by the typhoid bacillus, and the nature of the hemorrhagic exudate, remind us of similar phenomena observed in the lungs in the pestilential

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* *Lancet*, Mar. 16, 1898.

diseases" (Finkler, p. 217). I shall consider this more in detail in the section of this work devoted to the discussion of pneumonia as a complication of other diseases.

Next to the facts already spoken of, the question of a permanent disposition to pneumonia ought to be taken into consideration. This disposition is influenced by heredity, sex, and age.

Billard found pneumonia six times among seventeen patients who were born in a condition of extreme debility and emaciation. Grisolle found in a marasmic child born at the eighth month that the lower lobe of the right lung was changed into a tissue resembling liver, which was easily penetrated by the finger, and in certain places showed purulent infiltration.

If these facts seem to indicate a doubt whether croupous pneumonia was really present, this nevertheless could hardly be the case in an observation by Levy. The latter reports a case of intra-uterine transmission in which death occurred in the mother by a double pneumonia followed by suppurative pleurisy, while a pneumonia existed in the child, which from its anatomic character must be considered as a hemorrhagic catarrhal variety, with lobar distribution. The fact that the same disease was present in both mother and child was proved by the similarity of the organisms,—Diplococcus pneumoniæ of Fraenkel,—which was demonstrated as such with certainty by microscopic examination, cultivation upon agar, and successful inoculations in mice. The pneumonia of the child, judging from the anatomic finding, had existed, at the most, three days. The child, however, died forty-nine hours after birth; the infection, therefore, could not have taken place in any other locality than in the uterus, notwithstanding bacteriologic and microscopic examinations of the placenta were negative. But the fact that no bacteria were found in the placenta does not prove, by any means, that they were not transmitted from the mother to the child. Without becoming deposited in the placenta itself they may have been transmitted by the small hemorrhages that occurred in the placental circulation and very likely caused the premature labor. In support of this view are the following facts: (1) Seven hours after the birth of the child râles could be heard over the right anterior portion of the thorax; (2) examination of the blood of the child, which was obtained with a sterilized syringe under the customary precautions, from the left ventricle and the right lung, showed such a large number of cocci that infection by way of the blood channel seemed to be established without a doubt.

In addition to this, it is worthy of mention that Netter could regularly demonstrate in rodents the transmission of Diplococcus pneumoniæ from mother to fetus; and once in the child of a pneumonic woman, who died five days after her delivery, he observed pneumonia of the right upper lobe, pericarditis, pleuritis, peritonitis, meningitis, and otitis media, and in all these exudates, as well as in the blood, demonstrated Diplococcus pneumoniæ of Fraenkel.

The frequent occurrence of repeated pneumonias in children is significant and indicates that there is a congenital predisposition. Among 201 pneumonic children v. Ziemssen found 19 who had frequently had pneumonia; 14 had it twice, 3 of them three times, and 2 four times. I myself have seen a boy recover from three attacks of pneumonia with typical febrile crises between the eighth and thirtieth months; in another boy two attacks between the first and second years. It appears to me, however, that it is not justifiable to draw from this the conclusion that, once having withstood the disease, the disposition to a repeated similar attack of the same or other portions of the lung is increased, and that such a disposition occurs particularly in weak children.

Also in adults pneumonias may occur repeatedly. Very likely this depends upon a previously existing predisposition. But it is no more to be accepted in adults than in children that having withstood the disease once creates a predisposition.

Andral and Chomel showed the frequent occurrence of pneumonia in one and the same person. Among v. Grisolle's 174 patients 94 had recovered at some time previously from pulmonary inflammation which had occurred from one to eight times. In most cases a period of three to five years elapsed between the individual attacks. According to Riesell's experiences, among every hundred pneumonias almost regularly 50 represent first attacks, 32 second, 15 third, while the remaining 3 represent a fourth, or even more frequently repeated attack. Möllmann treated 944 pneumonias among 832 persons. Eighty-six persons—that is, 10%—had repeated attacks, 65 two attacks, 16 three attacks, 3 four attacks, and 2 five attacks. [Nineteen of the 195 cases I have to report had had previous attacks; of these, two had had two previous attacks and one had had three previous attacks. Norris found a history of previous attacks in 57 out of 500.—Ed.]

Whether sex has any influence upon the frequency of the disease appears to me, in spite of my own statistics and those of others, not yet to be established with certainty. If it were true that sex did have an influence upon the frequency of the disease, it would have to be assumed that there was a predominant morbidity in the male sex. In the Policlinic of Würzburg among 565 cases there were 85 women affected to every hundred men, and in the Policlinic of Kiel 75 females to every hundred males. According to Möllmann's observations of cases of pneumonia occurring in private practice, 579 occurred in men and only 365 in women; that is, 61.3% in men and 38.7% in women. These figures do not, however, prove anything with certainty as long as their relative percentage to the general morbidity of both sexes is not determined. But as the general sick-rate is not yet available, we will be obliged, in the absence of more accurate observations, to preliminarily confine ourselves to hospital figures. If we follow the only correct method,—namely, the determination of the relation of the men and women treated for pneumonia to the total number of admissions of both sexes,—and have a large number of patients at our disposal for this purpose, it can be expected that an approximately accurate result will be obtained. From January 1, 1880, to April 1, 1896, 1501 cases of pneumonia were admitted as indoor patients at the Altstädter Hospital in Magdeburg, among which there were 1223 men and 278 women. The total number, therefore, shows a percentage of 81.04% of men and 18.06% of women. Within the time above mentioned there were admitted altogether 36,540 patients, among whom there were 19,887 men and 16,653 women. The relation of all pneumonias to the total number is 4.1%: the pneumonias in men as related to the total number of men admitted, 6.01%; and in the case of the women, 1.7%.

According to this, the disease must be considered as occurring 3½ times as frequently in men as in women. [Norris' series showed males 382, females 118, out of 500, or about 3¼ times as many men as women.—Ed.]

It has, however, not been shown that there is any particular disposition connected with sex. The less frequent occurrence of the disease in women may depend upon the fact that, on account of their vocation, they are not as frequently exposed to heavy exertions in unfavorable weather as the men. This view finds support in the fact that wherever the women perform the same work as the men the frequency of the disease is equal in both sexes. Unfortunately, however, we possess but few observations in this respect. The observation of Rulf seems to be reliable. He observed in a region where both sexes were occupied in the same vocation (in the vineyards) that pneumonia seemed to be divided about equally among both sexes (44 men to 50 women).

The more frequent occurrence of pulmonary inflammations in men, therefore, is due to occupation.

The fact, however, that women contract the disease less than men seems to contradict the assumption that an indoor life, particularly in poorly ventilated rooms, is to be considered as of particular significance for the occurrence of the disease. If this were the case, more women would contract the disease than men.

The preference of pneumonia for a particular sex could be shown with more certainty if differences in children were observed. Unfortunately, however, there are very few reports concerning this, although those that do exist are not without value. Damaschino states that pulmonary inflammation occurs more frequently in boys than in girls. Among 16 cases, 12 were boys and 4 girls. In my observations I can show, against this small number, the positive fact that among my 1501 patients there were, between the ages of five and ten, 22 boys and 24 girls. Among those aged five years and less there were 35 boys and 22 girls. Even if we take all the cases up to ten years, there were 57 boys to 46 girls, which is cause enough to assume that sex does not cause any particular difference, and that the differences in the frequency of occurrence in more advanced age cannot be traced to the influence of sex.

The fact that pneumonia occurs more seldom among the better situated classes I would like to discuss according to my own subjective observations. Definite statistics concerning this fact are not available. Those that are at hand, however, seem to indicate that this is the fact; at least, according to the report of v. Jürgensen, it appears that the mortality of the English army shows that at the different stations fewer officers died from pneumonia than ordinary soldiers. Out of 1000 soldiers there were 12 deaths, and among 1000 officers but 5 deaths. The less frequent occurrence among better situated people may be explained by the possibility of better preventive measures and more resourceful auxiliary measures, so that the consequences of fatigue and bodily exertion, as well as the damage done by exposure to the elements, and particularly the influence of the rain, causing cooling of the surface of the body, may be more successfully combated.

In order to settle the question of permanent disposition, the subject of age and its significance for the occurrence of pneumonia is to be considered, and it is to be investigated whether any age is more predisposed than another.

It is a well-known fact that pneumonia occurs at any age. It is particularly to be mentioned, however, that in children pneumonia does not occur as rarely within the first year of life as is often supposed. The fact is that the subjective symptoms of the disease are frequently so little marked that the disease itself can easily be overlooked. According to my own observations, in private as well as in hospital practice, I fully coincide with the view of Rilliet and Barthez, as well as that of v. Ziemssen, and can assert that pneumonia occurs during the first years of life with comparative frequency.

In the following review of the statistics on the subject the number of cases within the first five years of life may appear somewhat high, and the possibility might be suggested that the autopsies in the hospital showed lobar, but not croupous pneumonia, had not Fraenkel and Reiche given similar reports.

As far as the age of the patients afflicted with pneumonia is concerned, the total number afflicted was:

Males.			Females.		
Up to 5 years	.. 35;	*i. e.,* 3.0%	Up to 5 years	.. 22;	*i. e.,* 8.0%
“ 10 “	.. 22;	“ 2.0%	“ 10 “	.. 24;	“ 8.5%
“ 20 “	..256;	“ 20.7%	“ 20 “	.. 77;	“ 28.0%
“ 30 “	..372;	“ 30.4%	“ 30 “	.. 65;	“ 23.4%
“ 40 “	..229;	“ 19.0%	“ 40 “	.. 30;	“ 10.6%
“ 50 “	..172;	“ 14.0%	“ 50 “	.. 23;	“ 8.3%
“ 60 “	.. 69;	“ 5.5%	“ 60 “	.. 14;	“ 5.0%
Over 60 “	.. 68;	“ 5.4%	Over 60 “	.. 23;	“ 8.3%
Total	.1223		Total	..278	

[In our 195 cases the age was as follows:

0 to 10 years	26
11 “ 20 “	28
21 “ 30 “	48
31 “ 40 “	32
41 “ 50 “	36
51 “ 60 “	13
61 “ 70 “	11
71 “ 80 “	1
	195

In Norris' series:

	Number Attacked.	Number Deaths.	Mortality.
1 to 10 years	71	10	14.0%
10 “ 20 “	71	7	9.9%
20 “ 30 “	136	25	18.0%
30 “ 40 “	84	22	26.0%
40 “ 50 “	67	29	43.0%
50 “ 60 “	30	16	53.0%
60 “ 70 “	13	10	77.0%
70 “ 80 “	6	4	66.0%
80 +	1	1	100.0%

—Ed.]

The relative frequency of the occurrence in the given ages cannot, however, be deduced from these figures. There is still to be considered a comparison with the total number of patients of each respective year admitted. For this purpose I have divided all the patients admitted into the hospital from 1892 to 1895 into the various ages. Within the time mentioned 4114 men were admitted and 4723 women, a total of 8837; and of these, there were:

Males.		Females.	
Up to the 5th year	397	Up to the 5th year	382
“ “ 10th “	88	“ “ 10th “	102
“ “ 20th “	806	“ “ 20th “	1645
“ “ 30th “	996	“ “ 30th “	1646
“ “ 40th “	597	“ “ 40th “	441
“ “ 50th “	565	“ “ 50th “	207
“ “ 60th “	311	“ “ 60th “	133
Over 60 years	354	Over 60 years	167
Total	4114	Total	4723

During the same time (1892 to 1895 inclusive) 220 males and 101 females, a total of 331, were admitted on account of pneumonia; and of these, there were:

Males.		Females.	
Up to 5 years	18	Up to 5 years	11
“ 10 “	8	“ 10 “	13
“ 20 “	36	“ 20 “	21
“ 30 “	59	“ 30 “	19
“ 40 “	30	“ 40 “	14
“ 50 “	55	“ 50 “	11
“ 60 “	15	“ 60 “	5
Over 60 “	19	Over 60 “	7
Total	220	Total	101

The comparative percentage of those ill with pneumonia to those admitted, according to the age, is as follows:

Males.		Females.	
Up to 5 years	4.5%	Up to 5 years	3.0%
" 10 "	9.0%	" 10 "	11.7%
" 20 "	4.4%	" 20 "	1.3%
" 30 "	6.0%	" 30 "	1.1%
" 40 "	5.0%	" 40 "	3.1%
" 50 "	6.2%	" 50 "	5.3%
" 60 "	5.0%	" 60 "	4.0%
Over 60 "	18.6%	Over 60 "	4.2%

All that I attempt to deduce from these figures is that the morbidity in males is the same at any age between the tenth and the sixtieth years of life; that the morbidity is high after the sixtieth year in males; and that the latter contract pneumonia between the tenth and the thirtieth years of life three times as frequently as do females in the corresponding ages. Whether the morbidity between the fifth and tenth years is really as large as the above figures indicate, or whether these figures are not sufficient, as far as these ages are concerned, to permit of a certain deduction, later investigations must determine.

Furthermore, it is to be considered whether one portion of the lung or an individual lobe is more disposed to pneumonia than other portions. This question has already been answered by Chomel, in that the right lung is more frequently attacked by pneumonia than the left. Andral even contends that the right lung is affected twice as often as the left. Grisolle has made exhaustive observations in this respect. Among his 280 cases the seat of the pulmonary inflammation was 166 times on the right side, 97 times on the left side, and 17 times it was bilateral. Among 1430 cases compiled by other authors, 742 occurred on the right side, 426 on the left side, and 262 cases were bilateral. The right lung was affected, therefore, in 51.9% of the total number, and the left lung in 29.8%. [In our cases the right lung was affected in 55.8%; the left, in 23.67% of the cases.—Ed.]

Grisolle has paid particular attention to the relative frequency with which the upper and lower lobes were affected. They became diseased in the proportion of 3 : 4. This, however, varies in individual years. In the years 1836–40 the upper lobe was affected in more than one-third of the cases, and in the year 1837 hardly in one-fifth of the cases. Regarding the predisposition of the right side, he remarks that this is due neither to the position of the patient nor to the more transverse direction of the right pulmonary artery, as Lombard believes, but that it is only the greater volume of the right lung which here can be of any particular significance. According to Huss, among 2616 patients the inflammation occurred 1398 times on the right side, 834 times on the left side, and 384 times on both sides, the relative percentage, therefore, being almost that of Grisolle: 53 : 32 : 15.

The table on page 436 presents my 1501 cases, arranged according to age and sex. In addition to this table, there is still to be remarked that among the 112 cases, in which both lobes of the right lung were affected, the lower and middle lobes were affected 66 times, the middle and upper lobes 45 times, and the lower and upper lobes only once; furthermore, that among the 189 cases in which both lungs were affected, there existed, in 18, various combinations which deserve to be briefly reported, although they do not call for special comment. The following are the various lobes that were affected:

Both upper lobes	14 times.
Both lower lobes	85 “
Right upper lobe and left lower lobe	10 “
Left upper lobe and right lower lobe	5 “
Left upper lobe and right middle lobe	1 time.
Left lower lobe and right middle lobe	2 times.
Entire right lung and left upper lobe	5 times.
Entire left lung and right upper lobe	5 “
Entire right lung and left lower lobe	22 “
Entire left lung and right lower lobe	17 “
Entire left lung, right upper lobe, and right lower lobe	2 “
Entire left lung, right lower lobe, and right middle lobe	4 “
Left upper, right upper and middle lobes	1 time.
Left lower and right middle lobes	2 times.
Left lower, right lower, and right upper	4 “
Left lower, right lower and middle lobes	6 “
Left lower, right upper and middle lobes	1 time.
Both lungs totally	3 times (terminating fatally).

Among the 112 cases (7.5%) in which only two lobes of the right lung were affected, the—

Upper and middle lobes were involved in	45 cases.
Lower and middle lobes were involved in	66 “
Lower and upper lobes were involved in	1 case.

[Location of lesion—195 cases, Musser ; 500 cases, Norris—

Right lower	55 = 28.2%	145 = 29%.
Right middle	4	14 = 2%.
Right upper	19	55 = 11%.
Entire right lung	12	45 = 9%.
Right middle and lower	10	
Right upper and middle	7	
Right upper and lower	2	
	109 = 55.8%	
Right and left lower	14	60 = 12%.
Right lower and middle, and left lower.	1	
Right lower and middle, and left upper.	1	
Right upper and middle, and left upper	1	
Right middle and left lower	1	
Entire right lung and left lower	2	
Left lower, left and right upper	2	
Entire left lung and right lower	3	
Left and right upper	2	4 = 9%.
	27 = 13.8%	
Left lower	36 = 18.4%	115 = 23%.
Left upper	6	20 = 4%.
Entire left lung	4	35 = 7%.
Left side	46 = 23.6%	
Location doubtful	13	

—Ed.]

TABLE SHOWING THE INVOLVEMENT OF INDIVIDUAL LOBES IN 1501 CASES OF PNEUMONIA.

Age (Years).	Right Lower.		Right Upper.		Middle Lobe.		Right—Entire.		Right. Two Lobes.		Left Lower.		Left Upper.		Left—Entire.		Both Lungs.		Total.	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Five	4	2	5	2	0	0	6	5	3	1	3	7	3	1	6	2	5	2	35	22
	6		7		0		11		4		10		4		8		7		57	
Ten	3	4	9	6	0	0	0	1	0	0	7	9	2	4	0	0	1	0	22	24
	7		15		0		1		0		16		6		0		1		46	
Twenty	56	19	18	8	9	2	22	6	19	0	83	23	3	4	17	4	29	11	256	77
	75		26		11		28		19		106		7		21		40		333	
Thirty	91	15	31	3	11	2	41	7	34	1	81	16	12	1	23	6	48	14	372	65
	106		34		13		48		35		97		13		29		62		437	
Forty	57	6	17	5	7	0	16	5	28	1	56	5	13	0	15	2	20	6	229	30
	63		22		7		21		29		61		13		17		26		259	
Fifty	42	4	15	2	1	0	16	2	10	1	42	5	6	1	13	3	27	5	172	23
	46		17		1		18		11		47		7		16		32		195	
Sixty	13	5	11	0	2	0	9	4	5	2	16	1	4	0	1	2	8	0	69	14
	18		11		2		13		7		17		4		3		8		83	
Over sixty	15	4	8	5	0	0	11	0	4	3	16	3	1	2	4	2	9	4	68	23
	19		13		0		11		7		19		3		6		13		91	
Total	281	59	114	31	30	4	121	30	103	9	304	69	44	13	79	21	147	42	1223	278
	340		145		34		151		112		373		57		100		189		1501	
Percentage	22.6		9.6		2.3		10		7.5		24.8		3.9		6.7		12.6		100	

Regarding the relation of both halves of the lungs, it was seen that the right side was diseased 782 times alone, the left side 530 times alone, and both lungs 189 times; the relative percentage almost entirely coincides with that reported by Grisolle and Huss; it is 52% : 35.2% : 12.8%.

In addition to these figures, it is deserving of particular mention that such a careful observer as v. Ziemssen found that in children there was no difference whatever in the frequency of the disease on the right and left sides. According to his reports, among 191 cases of croupous pneumonia occurring in children 91 were on the right side, 88 on the left side, and 11 on both sides. This difference between children and adults leads me to assume that perhaps the work of adults, who use the right arm and the musculature of the right half of the thorax more than the left side exerts an influence upon the right lung, and makes it especially liable to contract the disease. It is still to be determined whether this influence is produced by means of the pulmonary vessels or through mechanical concussion of the lung tissue.

CLINICAL COURSE.

In healthy individuals who are not above middle age the disease generally sets in abruptly. In children a violent convulsion may occur at the onset, and in youthful individuals violent vomiting may occur, while in middle age an intense chill ushers in the disease. In all cases there also occurs a marked rise in temperature. Pulse and respirations are markedly increased, the face is flushed and occasionally livid. The patients complain of a feeling of intense depression, headache, and marked thirst. Frequently from the beginning "a stitch in the side" occurs. More rarely than is stated, however, does marked dyspnea occur from the onset. When it does occur, it is to be attributed to the pain in the side, which renders complete respiration impossible.

In the minority of cases a feeling of general malaise lasting several days precedes the onset; this is to be taken into consideration in the diagnosis and prognosis, and particularly as regards the outlook for complete recovery. We should be careful not to ignore the fact that an acute miliary tuberculosis of the lungs may have a similar onset.

When pneumonia is added to a disease already present,—in the puerperium, enteric fever, variola, delirium tremens, and psychoses,—it may begin without any characteristic symptom. The patients do not complain of pain; cough and dyspnea need not necessarily be present. Only the fever that was previously present rises higher. If the lungs are not examined, the disease may be entirely overlooked.

The short paroxysms of cough occurring with the onset of the disease gradually become more severe. They increase the pain in the side, and, in addition, give rise to pain in the head, which becomes so violent that the patients fear the paroxysms of cough, and when the latter occur, hold their heads with both hands. The pneumonias affecting the base are most frequently accompanied by a stitch in the side.

At first no secretion accompanies the cough. Later, however, generally even on the second day of the disease, there is a characteristic bloody sputum, which represents a very important symptom of the disease, and from the very beginning makes a diagnosis of pulmonary inflammation permissible. At least in an otherwise healthy individual, in spite of the preliminary absence of certain objective symptoms, one will not go amiss in making a diagnosis of "pneumonia" if one depends, on the one hand, upon the characteristic sputum, upon the violent fever, as well as upon the stitch in the side, provided, on the other hand, cardiac disease, as well as diseases giving rise to emboli in the lung, can be excluded. But this does not by any means mean that a pneumonia cannot occur where cardiac disease and even emboli already exist.

The characteristics of pneumonic sputum consist in its tenacity and its bright red appearance. Where infarcts exist, even when these affect a whole lobe of the lung, the expectorated blood is of a dark red color. The sputum expectorated in croupous pneumonia has correctly been called "rubiginous" ("rusty"). The expectoration in pneumonia may of course present other appearances. During the first two days but a few streaks of blood may be mixed with the sputum, which adheres to the cup, and it may, in the further course of the disease, assume a lemon yellow, greenish, or darker hemorrhagic prune-juice appearance, and in the latter case become thin and watery. Generally in those cases where the sputum is needed for purposes of diagnosis it is entirely absent; this is the case in pneumonia affecting the upper lobes, in delirium tremens, and in pulmonary inflammations which occur secondary to other diseases.

The most characteristic symptoms are obtained by objective examination, by percussion and auscultation. Independent of every subjective symptom, at the beginning of the disease a somewhat empty, high percussion note points to the diseased side or lobe. This change is to be attributed to the diminution in size of the alveolar lumen, to the swelling of its epithelium, and to the marked filling of the alveolar capillaries. The decrease in the elasticity of the lung tissue in the diseased portions which is associated with these phenomena, contributes to confine the vibrations to a smaller area.

The decrease in the elasticity of the pulmonary tissue may occasionally give rise to a tympanitic percussion note and occasion doubt as to whether one is really examining the diseased side. In pneumonias lasting one to two days it may even happen that one cannot know which the diseased side is. Even the presence of stitch in the side does not always decide the question, for it may occur upon both sides, or on the side opposite to the diseased lung. Even auscultation does not necessarily elicit characteristic signs at the beginning. The breathing may simply be weakened. Generally, however, the characteristic crepitant râles soon occur, which, together with the above-named symptoms, clear up the diagnosis.

In the further course of the disease, frequently even on the second day, percussion shows complete dulness; and auscultation reveals bronchial inspiration and expiration, generally without râles. Vocal fremitus is increased, bronchophony is heard, and what, according to my experience, may be considered as the most important point of differentiation from the bronchophony and egophony heard in pleural effusion, is a soft bronchial terminal respiratory sound heard during speaking after each word, and occasionally also after each syllable, corresponding to the expiration.

If these signs were always present, the diagnosis would give rise to no particular difficulties; but variations are frequent.

In the first place, in pneumonias of the upper anterior lobes there is rarely well-marked dulness, which may also be said of pneumonia in the same position occurring posteriorly; the same is also true of bronchial breathing. The sputum is rarely bloody. The reason is due to anatomic conditions. The gray hepatization which is preceded by hemorrhage into the alveoli is rarely so copious in pneumonia of the upper lobe that the alveoli are completely filled.

On the other hand, pneumonias which complicate other affections are frequently characterized by well-marked dulness.

In the further progress of the affection, especially in the stage of transition from red to gray hepatization in cases that run a favorable course, the symptom-complex which is noted during the earlier course of the disease is rarely changed. The high fever, which is marked from the onset of the affection, only shows morning remissions; respiration and pulse frequency are increased; the skin is hot, usually dry; only rarely and very transitorily is slight perspiration noted. Occasionally herpes is added to this. The attacks of cough are frequent, but not of long duration. Rusty sputum or tenacious mucoid sputum is brought up in lessened quantity. The stitch in the side, which is endurable during inspiration, becomes exceedingly painful during the attacks of cough. Both contribute to interrupt sleep. The appetite is completely lost; only marked thirst is present. The tongue is for the most part thickly coated. The urine is secreted in lessened quantity; the bowels are constipated. The nervous system shows its sympathy in the form of mild delirium.

This for the most part normal course may be interrupted by many dangerous phenomena. To these belong, in the first place, conditions of collapse. Whether these are due to a disturbance of the function of the heart has not yet been proved. Probably thromboses in the pneumonic areas are more frequent than has been supposed. Emboli make their way into the right ventricle, and from there into the pulmonary arteries of other previously healthy portions of the lungs, and, through the sudden interruption in a large circulatory area, bring on symptoms of collapse. If the emboli are small, deleterious effects do not necessarily follow.

Furthermore, serious symptoms on the part of the nervous system may arise. Severe hallucination, delirium, and, in drunkards, delirium tremens, disturb the otherwise favorable course.

In spite of all this, the outcome may even be favorable. On the fifth, seventh, or ninth day the crisis occurs, with a complete fall of the temperature to normal. In a lesser number of cases the temperature falls in the course of several days by lysis.

With the beginning of crisis the objective phenomena undergo a rapid change. The percutory and auscultatory phenomena return to normal in the same manner as they had progressed from normal conditions of the pulmonary structure to hepatization. The intense dulness diminishes, the note becomes louder, then hollow and high, until, with or without a tympanitic admixture, it changes into the normal full and deep pulmonary resonance. Bronchial breathing as well as bronchophony and the increased pectoral fremitus disappear; crepitation is noted, and may or may not be accompanied by medium-sized râles, until finally normal vesicular respiration returns. This recession of the objective phenomena may be completed within twenty-four hours. Just as rapidly all subjective symptoms subside; and the cough may even disappear suddenly.

When the affection terminates by lysis, a slower disappearance of the objective and subjective phenomena is noted. Under these circumstances a careful examination of the temperature curve is especially necessary. Any irregularities in the expected fall in the temperature, which ought to show a remission of the morning and evening temperature as compared with the temperature of the preceding morning and evening, must be looked upon with suspicion, because during this time, as well as at the acme of the affection, the inflammation may attack other previously healthy parts of the lung, as may easily be verified by examination, or a serous and occasionally purulent pleurisy may have been added which corresponds to the position of the affected pulmonary lobe, and which may lead to the erroneous idea that one is dealing only with delayed resolution. If, therefore, several days after the beginning of lysis dulness is noted over the affected lower lobe,—because the base is exclusively affected in such cases,—or if the dulness persists during the course of irregular temperature remissions, whereas the other parts of the lung do not show evidence of disease, or even if dulness with a normal temperature is noted, the suspicion must arise that a pleural effusion has been added to the previously existing pneumonia. The demonstration of pectoral fremitus, which is valuable in the diagnosis of this condition, is frequently impossible, especially in women and in those patients that are hoarse or that suffer from aphonia. In these cases, if dulness upon percussion should last some time, or if the course of the affection appear serious, aspiration is absolutely necessary. In the case of a woman in whom dulness existed after several days' complete absence of fever, and in whom the objective phenomena did not give sufficient reason to suspect a pleural effusion, I found by aspiration the existence of a very large empyema, and by instituting treatment this condition was relieved and the case terminated in recovery.

Death rarely, perhaps one might say never, occurs in croupous pneumonia in the first two days. Should death occur in this time, it is probably not due to a croupous pneumonia, but always to an atypical pneumonia produced by another kind of infection.

Wide distribution of the pneumonic lesion appears to be relatively the least ominous factor. Pneumonias which have only affected a lower lobe may give rise to as many dangers as those which affect an entire lung. Of course if the respiratory area is reduced below a certain point, the danger incident to the disease is thereby enhanced.

Of the utmost importance to the outcome of the affection are the toxic substances conveyed from the lungs to the other organs. Many pneumonias which run a fatal course cannot be explained otherwise. Death occurs, with a very rapid small pulse, frequent respiration, cold extremities, and nervous symptoms giving rise to more and more marked coma. The cardiac symptoms merely form a part of a general intoxication. That the height of the fever has a damaging influence upon the muscle of the heart is not to be supposed. For, in the first place, in other diseases equally high temperatures, lasting a much longer time, occur without producing such effects upon the heart; and, in the second place, in those cases in which death occurs from cardiac weakness, the temperature need never have reached a very high point.

In other cases which run a fatal course, the increase in the number of respirations and the presence of ronchi up to the time when pulmonary edema develops, show that the phenomena which threaten the patient's life are referable to the lungs. The edema of the lungs which arises under these circumstances is very likely due to a spreading of the pneumonic process which no longer is able to fully develop itself, but under the influence of weakened cardiac action shows itself in this manner. A considerable part in the fatal termination is also due to the localization of the diplococcus in vital organs, in the heart, and, what may occur much more frequently, in the meninges [and in the colon.—Ed.].

Further on I shall come to speak of some rarer causes which give rise to the fatal issue; here may only be mentioned that occasionally sudden death occurs during or after the crisis from the patient suddenly assuming the erect posture. Oligemia, according to Bollinger, is the principal cause of this fatal issue in pneumonia.

ANALYSIS OF SPECIAL SYMPTOMS.

The real beginning of the disease—that is, the onset from the time that the effects of Diplococcus pneumoniæ upon the parts of the lung which have previously been predisposed are manifested—can rarely be definitely determined, as in most cases a brusque onset of the phenomena of the affection without symptoms or with the gradual onset of the deleterious conditions, or, as we are apt to say, the prodromal stage, is entirely absent. Probably milder disturbances of the bodily functions are overlooked; frequently enough the physician himself, when he notes the stormy symptoms of a pneumonia, entirely forgets to ask about premonitory symptoms. Such a careful investigator as Grisolle did not allow the observation to escape him that in almost a quarter of all cases (50 out of 205) prodromes existed, most frequently in persons between the ages of thirty and forty, as well as between the ages of fifty and sixty. Loss of appetite, malaise, pains in the back, and chilliness are noted. In 19 of the previously mentioned 50 cases a slight bronchitis preceded the affection. [This is entirely in conformity with our experience. —Ed.] In the rarest cases the prodromes last longer than five days. Traube says that in the development of pneumonia in some cases prodromes exist which have a duration of from several days to four weeks; the patients complain of muscular weakness, of abnormal sensations, such as headache, pains in the extremities; of disturbances in the digestive apparatus, loss of appetite, etc. After these prodromes have lasted a longer or shorter time a chill occurs, which is followed by the development of the local phenomena of the pneumonia. Occasionally the pneumonia arises in the form of a fever, which exists for several days without any symptoms referable to the respiratory apparatus. During this time no abnormal acoustic signs, nor any cough, expectoration, or abnormal sensations in the respiratory apparatus, are noted. Knövenagel has demonstrated, by carefully questioning his patients, that in some cases symptoms of the disease had existed only three or four days before their admission to the hospital; although in the majority of cases symptoms were noted seven to nine days previously. Penkert gives the period of incubation as from five to eight days.

Of especial value in the determination of the period of incubation are those cases in which contusions are the cause of the disease. Here the time between the contusion and the onset of the chill, although it varies in individual cases, is, in general, brief. According to the statement of Stern, only the bloody expectoration which is frequently present at the onset is a symptom which departs from this rule, whereas the other symptoms do not differ from the usual symptoms of pneumonia. The chill, however, frequently occurs after a few hours [in our case, within the twenty-four hours.—Ed.], sometimes in from one to two days after the contusion.

The attempt to explain the existence of the prodromes, and the period of incubation which runs its course without obvious prodromes, can only be done by the assumption that, step by step with the local changes, materials are accumulated in the blood whose quantity gradually increases until either a chill or convulsions (in the case of children) or vomiting shows the full development of the affection. The three last-named, apparently very different symptoms, speak in a conspicuous manner for the justice of the assumption that through the blood materials are brought to the brain which are capable of producing an irritation there, and also that these symptoms are referable to a cerebral disturbance. No other comprehensive explanation—and such a one would only be admissible—has ever been offered.

The chill, the convulsions, and the vomiting are always followed by a rise in temperature. These symptoms then determine the time from which the beginning of the affection is determined, and from which the course and duration of the affection are reckoned. Von Ziemssen, Traube, and v. Jürgensen advise, quite properly, that the duration of the affection should not be counted by calendar days, but by periods of twenty-four hours from the hour of the onset.

The anatomic changes in the pulmonary structure can only be determined objectively after the onset of the previously mentioned symptoms.

More difficult is the determination of the onset of the affection when—as rarely happens—frequent attacks of chills precede and no objective symptoms relating to the respiratory organs can be determined after the first chill. These repeated chills I have seen in cases in which there was great weakness of the myocardium previous to the onset of the pneumonia.

The chill and the other coordinate symptoms may be entirely absent in the beginning of pneumonias occurring in the course of delirium tremens or during the course of other acute and chronic affections in elderly people. In aged persons, according to Hourmann and Déchambre, the disease begins acutely in only one-half of the cases. In the remainder the affection shows itself by malaise, weakness, increase in pulse frequency, and fever. [Particularly the weakness and increased pulse-rate.—Ed.]

Is the action upon the brain of the poison circulating in the blood in such cases less intense? or does the influence of the affected brain show itself less markedly because the vascular system under these circumstances is less powerful? I believe the latter is the more likely.

The onset by chill is by far the most frequent. Louis noted chills 61 times in 79 cases; Grisolle 145 times in 182 cases; 110 times chill was the very first symptom; in the remaining 35 chill arose only after the stitch in the side, shortness of breath, cough, and vomiting had occurred. [It was noted in 92 of our series of 195 cases, absent in 56, and not noted in 47. It was noted in 250 of Norris' series.—Ed.] The duration of the chill varied. Twice the chill lasted nine hours; in nearly one-half of those (17 out of 37) that had no chill the affection arose during the night; the warm body was more protected from the influence of the outer temperature. Chill is also more frequently absent in autumn and summer than in the spring and winter. In children also it is rare.

Especially characteristic of pneumonia is the rapid rise of the tem-

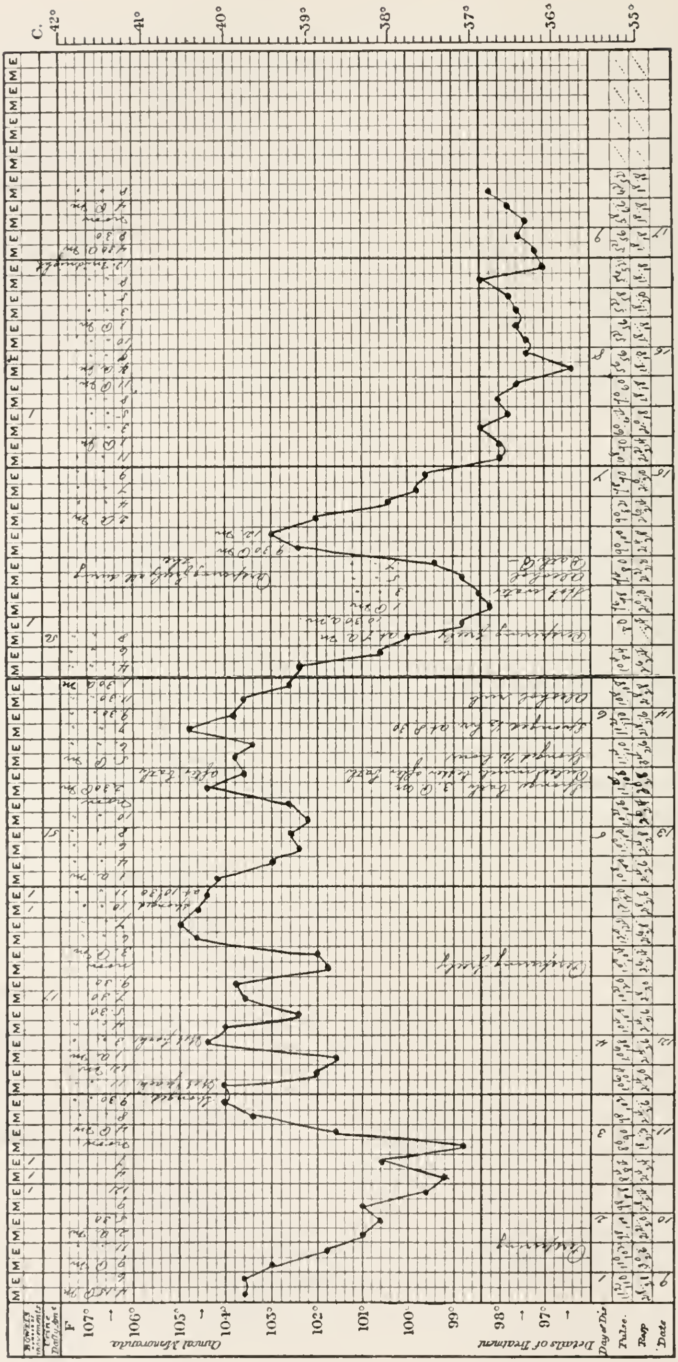

FIG. 25.—Typical course of fever in pneumonia with pseudocrisis and crisis.

perature after the chill. Von Ziemssen, Thomas, v. Jürgensen, and Finkler have given convincing proofs of this. Von Jürgensen noted: (1) In the case of a girl seven years old, three hours after a chill and violent vomiting, the temperature was 41.1° C. (106° F.); (2) in a nine-year-old boy who was taken ill with marked cerebral phenomena, four hours after the onset 40.4° C. (104.7° F.); (3) in a fifty-six-year-old woman, eight hours after the chill 40.2° C. (104.4° F.). Finkler found an hour and a half after the chill in a forty-year-old man 40.6° C. (105° F.). In general the temperature rises in a few hours above 39° C. (102.2° F.), and even after that continues to rise until it reaches a height of nearly 40° C. (104° F.). In severe cases even 41° C. (105.8° F.) and over may be reached. "If women are taken ill of pneumonia during the time in which their menstruation should occur, very high temperatures are usually noted" (v. Jürgensen, p. 57).

The further course of the disease is characterized by continued fever (see chart, Fig. 29); that is to say: "Except during the periods of rise and fall the temperature curve is composed of the normal curve *plus* an addition of *x* degrees. This increase in the temperature remains constant; the normal curve shows the daily variations, and within certain limits deviations from these occur upon special days" (v. Jürgensen, p. 55). Lebert says, on the other hand, quite properly ([145], p. 25): "An absolutely continuous fever does not occur in pneumonia, and we may only speak of such, in which the morning remissions are slight and do not exceed ½° Celsius (see chart, Fig. 26). But even in these cases the subcontinued course is not regular, since before as well as after, and between lesser differences, a rise which amounts to one degree or even more frequently occurs, thus changing the subcontinued course into a slightly remittent type. [We have observed it to be remittent the first two or three days in many cases.—Ed.] Small differences are noted in mild as well as in severe cases; especially in the latter the average slight remissions alternate not only changed with greater ones, but with the intercurrent drops, and these intense protracted cases not rarely later terminate by lysis."

Besides this, a well-marked remittent type also occurs in which daily differences of from 1° to 1.5° C. (1.8° to 2.7° F.) even 2° C. (3.6° F.), may occur. According to Lebert's experiences, such constituted curves belong to the cases in which a favorable prognosis may be given, and this zigzag appearance of greater exacerbations with marked remissions has no unfavorable significance. (See chart, Fig. 26.)

In the course of an intermittent temperature, a pyrexia and a rise alternate in a pretty regular rhythm. This form in its complete development is only noted during the time of intermittent fever epidemics. It terminates, either so that after a defervescence no new rise in temperature occurs and convalescence follows, or after several recurrences the intermittent character disappears and the pneumonia with a relatively high temperature then terminates by lysis like a remittent form (Wunderlich [261], p. 366).

A relapsing course of the fever was formerly noted after the use of general or copious local bleeding. It can, however, occur without any intervention of this kind. A rapid defervescence usually occurs early, even upon the second or third day. The temperature may then remain normal for thirty-six hours or longer; but one does not note a proper involution of the local changes. Suddenly the temperature again rises very rapidly, but usually does not quite reach the former height. It remains but for a few days at the fastigium and then a definite defervescence occurs, or the fever rises a second, and sometimes even a third time (Wunderlich, p. 365). (See chart, Fig. 25.) An analogous course of the temperature is described by Bertrand. He calls such cases "*pneumonies à poussées successives.*"

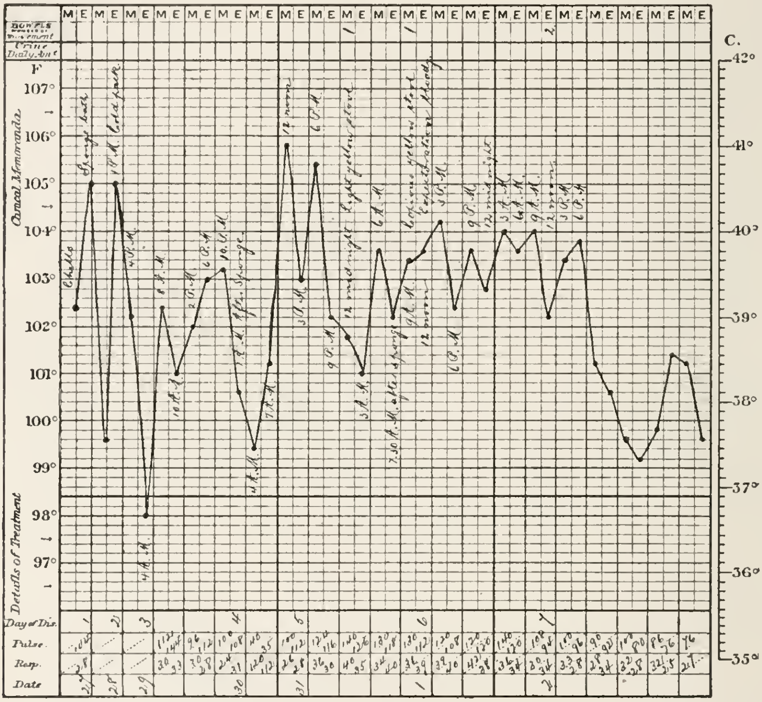

FIG. 26.—Remissions of fever during first five days. Doubtful response to cold pack, which was administered once only.

Wunderlich noted a temperature course with recrudescing fastigium in cases in which, after hepatization of a part of the lung, a second lobe or the other lung was attacked. The temperature, which had previously been favorable, or moderately favorable or was about to enter on a defervescing course, suddenly presents a sharp rise, which is then followed by a continuous or a subcontinued course.

The final fall in the temperature may come at the end of a period of gradual decline; or the temperature may be persistently high up to the last day of the fever; or the final defervescence may be preceded, on the day before, by a pseudocrisis (see chart, Fig. 25) [14 out of 195 cases, and 54 in a total of 500.—Ed.] in which the temperature goes down to normal, and which is followed in favorable cases, according to my observations, by a brief, moderate, final exacerbation; or, finally, a marked rise in the temperature (*perturbatio critica*) may take place immediately before the beginning of the final defervescence (see chart, Fig. 27) [Case of B ——, in which the temperature rose to 107° F., but fell to 99° F. in five hours.—Ed.]. Whether the occurrence of such an excessive rise toward the end of the attack is a sign of impending death cannot at once be positively determined, even after due consideration of all the other symptoms. If severe nervous symptoms precede death, a rapid rise in the temperature to 41° C. (105.8° F.), or even 43° C. (109.4° F.), may occur (Wunderlich, 358).

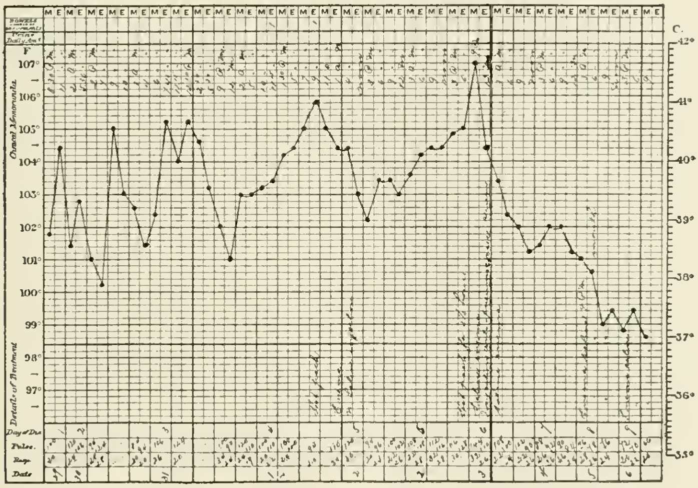

Fig. 27.—High temperature with pre-critical rise.

An enormous rise in the temperature may also occur immediately after death (Lebert).

The communication of Traube in respect to the important part played by crises and critical days leads to important discussion. Upon the basis of his experience he notes that on the fifth, seventh, ninth, or eleventh day in the course of an acute attack, a drop in the temperature not rarely occurs spontaneously or suddenly, which, however, does not necessarily immediately lead to convalescence, but gives rise to a marked and permanent lessening of the fever. He has never noted such a condition on any of the days between those stated. The sudden falling of the temperature which leads to convalescence, if it occurs, as it generally does, before the fourteenth day, is always noted upon the third, fifth, seventh, ninth, or upon the eleventh day of the disease.

Only a partial confirmation of this view is given by v. Ziemssen.

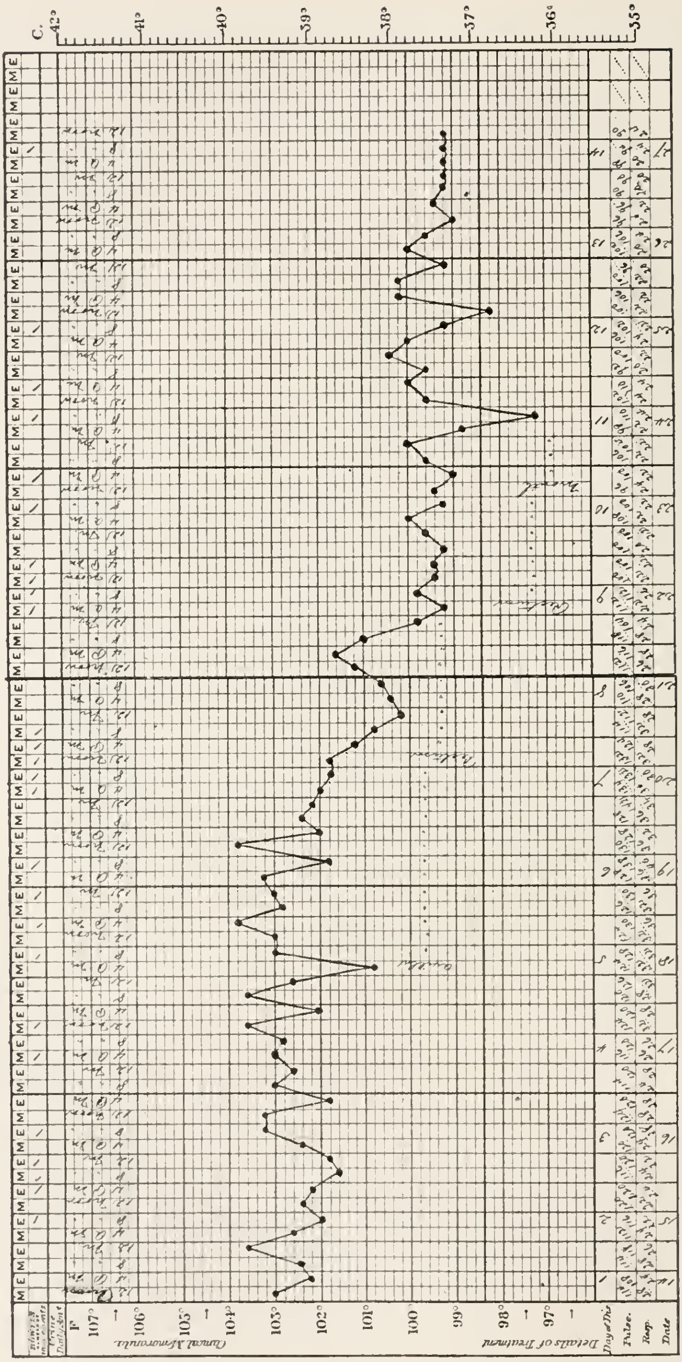

FIG. 28.—Typical course of fever in pneumonia except termination, which was by lysis. Toxemia great, though fever moderate.

Among 107 cases of pneumonia, the crisis began twelve times upon an even, 95 times upon an odd, day of the disease. Wunderlich, on the other hand, found among 75 cases the crisis but 45 times upon an odd day and 30 times upon an even day of the disease. Just so Thomas, after an exhaustive investigation, arrived at the conclusion that the number of cases in which the crisis began upon an odd day was not greater than the number in which the defervescence was noted upon an even day of the disease. Treatment had absolutely no influence upon this condition.

Liebermeister, upon the basis of statistics of 824 cases, which were collected from various well-known authors, brings the proof that among 817 of these cases, in 374 the crisis occurred upon an even day, and in 443 upon an odd day of the disease. This, nevertheless, is slightly in favor of the odd day of the disease.

In regard to the frequency of crises as well as critical days, the analysis of all of my cases shows 873 complete crises in 1501 cases, or 57.5%. [In our series the crisis occurred in 46.2%; in Norris' in 60.2%.—Ed.] The critical days were:

Day.	Men.	Women.	Total.	Norris, Crisis in 500 Cases.
1	.	1	1	0
2	2	.	2	3
3	51	10	61	1
4	40	13	53	4
5	96	27	123	12
6	107	9	116	34
7	129	26	155	43
8	86	9	95	58
9	109	18	127	42
10	42	8	50	30
11	37	13	50	18
12	9	.	9	13
13	14	2	16	8
14	6	.	6	5
15	1	.	1	3
16	2	1	3	5
17	1	.	1	5
18	1	1	2	4
19	1	.	1	2
20	1	.	1	11 (or after)
Total	735	138	873	

The crisis therefore occurred on odd days in 536 cases; on even days in 337 cases. [In Norris' series the crisis occurred on odd days in 139 cases; on even days in 162 cases.—Ed.]

It is also worth mentioning that among the 22 cases of pneumonia in children up to the fifth year of life which ended critically, the crisis occurred after the seventh day in 11 cases; among 150 adults over forty years of age the crisis occurred 73 times after the seventh day, while in the rest, between the fifth and fortieth years of age, in 699 pneumonias the crisis only occurred 273 times after the seventh day; therefore here in one-third, whereas in the first years of life, and in the later years of life in one-half the cases, the crisis occurred after the seventh day. (See chart, Fig. 25.)

In reference to the time of day at which the crisis occurs, Wunderlich says quite justly that in the majority of cases it occurs in the late evening hours, occasionally even in the afternoon or at night, comparatively rarely in the morning and during the mid-day hours. The defervescence is often so rapid that in spite of the high temperature the normal level may be reached from evening until morning. In a case reported by v. Jürgensen ([117], p. 59) in which the temperature was taken every five minutes, a fall from 40° to 37.8° C. (104° to 100° F.) took place in five hours and twenty minutes. Very frequently, however, the critical fall requires between twenty-four and thirty-six hours. The temperature then falls steadily; that is, without an evening rise.

The temperature may also fall below the normal, and in some few cases remain for a day or two subnormal.

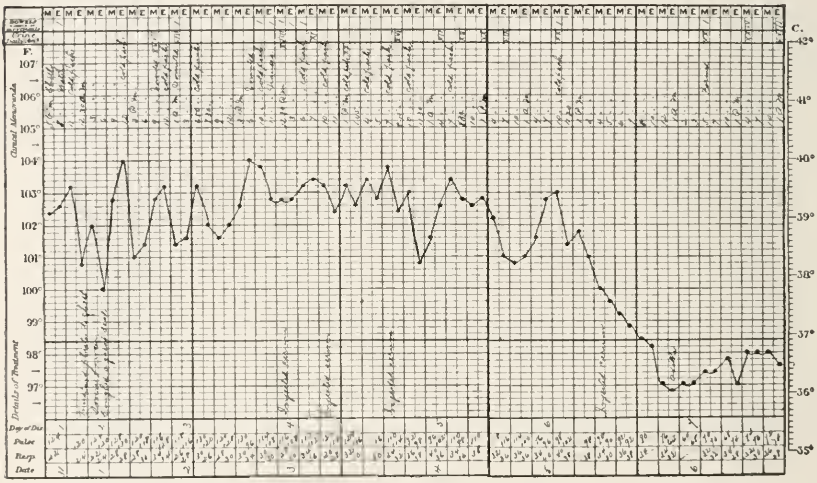

Fig. 29.—Typical course of fever in pneumonia.

In some cases no critical fall is noted, the temperature reaching the normal by lysis (see chart, Fig. 28) (Wunderlich, p. 219), and either in a continuous or slow process of falling in which the temperature from morning to evening falls less than from evening to morning, or becomes remittent in that the morning remissions alternate with decided evening exacerbations.

It is well in all cases in which the drop in temperature occurs by lysis to look for the existence of a complication. Even if it is not proper in such cases to exclude a true pneumonia and accept pneumonic relapses as the most frequent cause, nevertheless complications may be the cause of a fall in temperature by lysis. (See chart, Fig. 30.) For example, a fresh part of the pulmonary structure being invaded by the pneumonic process, or the addition of small pleural exudates, which in a short time may spontaneously disappear. Wagner believed that termination by lysis is more frequent at some periods than at others. At one time he saw them more frequently than formerly. In the years from 1881 to 1883 he saw among 199 pneumonias only 115 terminate by crisis (26 by protracted crisis) and 84 by lysis.

Pneumonia runs its course without fever in the aged. Hourmann and Déchambre say: "Old women do not even complain of being ill. No one in the ward notes the slightest change in their condition. They get up, make their beds, go around, eat as formerly, then they feel themselves somewhat weak, lie down in bed, and die. One finds upon postmortem a large part of the parenchyma of the lung in a condition of abscess."

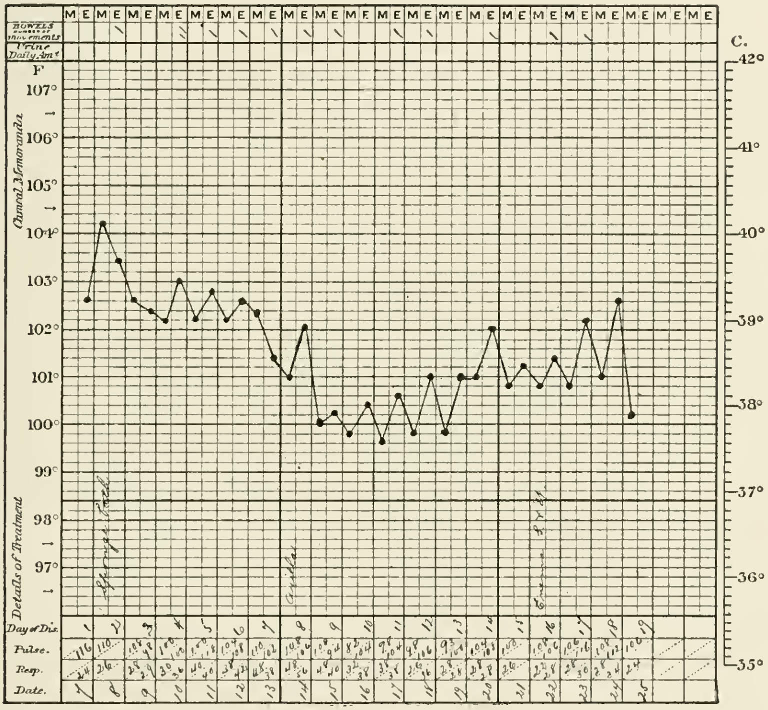

FIG. 30.—Usual course of temperature followed by empyema, disclosed by physical signs the sixteenth day.

In close relationship with the fever is the decomposition of nitrogen, or the change in body-weight. By means of the very careful investigations of Hubbert and Riessell in reference to the decomposition [catabolism?] of nitrogen in a person affected by croupous pneumonia the proof is brought that just in this disease an enormous oxidation of organic albumin occurs, which is in relation to the height of the temperature. They determined that the person affected, a man aged twenty-five, in five days of fever took on 83.35 gm. (2.68 ounces) nitrogen, that is, flesh containing 3.4% nitrogen; but during the same period he lost 2.45 kg. (5.40 lbs.), or 10.2%, of his entire body-weight. In estimating the accumulation of nitrogen from the sixth to the tenth day as 89.54 gm. (2.87 ounces) nitrogen, or 2.65 kg. (6 lbs.) flesh, the loss during the five days of illness amounted to 21.2% of his weight.

These results correspond to the astounding loss of weight which occurs in pneumonia in the course of but a few days. As it is my rule to weigh the greater number of pneumonia patients upon their admission to the hospital, also after the crisis, and finally after their discharge from the hospital, I have been able to determine that patients weighing from 130 to 150 pounds frequently lose after the crisis 20 pounds of their body-weight. But this loss is very rapidly made up. After discharge from the hospital the patients frequently weighed considerably more than prior to their admission. It must, however, be noted that the patients had already suffered some loss of weight, due to the disease existing before their admission to the hospital, as they were often ill for some days before being admitted.

If we now direct our attention to the **local symptoms,** and especially those relating to the respiratory organs, the most prominent phenomena which arrest our attention are **pains** or "a stitch in the side." These belong to the most frequent symptoms of pulmonary inflammation, and are scarcely ever absent when the lower lobes are involved, especially when persons in middle life are affected, who are overcome while previously healthy. This stitch in the side is rarer in pneumonia of the upper lobes, and in the pneumonia of childhood; occasionally, however, the opposite is true, and the children suffer from very severe pain lasting for some days. It is rare for aged persons to complain of these pains; they are almost never felt or mentioned by delirious patients.

Usually the pain is confined to a limited area and is scarcely ever widely distributed. Among the 301 patients of Grisolle's these pains were absent but 29 times, therefore in but one-tenth of the cases. Of the 182 patients, 161 had these pains in the first twelve hours. Of the remaining 21, 17 noted the pain at the end of the first day, 4 between the second and fourth days. The pain corresponds to the inflamed pulmonary area. In double pneumonia pain was complained of on but one side. Pressure upon the affected intercostal space increases the pain. This does not contradict the statement that occasionally the patients are able to ameliorate the pain by pressing the hand over the affected area. In this manner circumscribed pressure is not exerted, but the expansive force of the affected part in the thorax is restricted. [In 56, or 22.1%, of our series pain was marked. Twenty of these cases had easily defined pleurisy.—Ed.]

In reference to the seat of the pain, we must thank Grisolle for exact statistic reports. In 173 cases the pain was in the region of the nipple 89 times; at the base of the lung and at the upper extremity of the seventh or sixth rib, spreading from below upward, 39 times;

in the side and from ten to twelve centimeters below the mammillary line, 13 times; anteriorly and laterally, 10 times; inside of the nipple, toward the border of the sternum, 6 times; posteriorly, especially in the fossa infraspinata, 5 times, and in the hypochondrium 4 times; in the axillary space, twice; in the region below the mammilla, twice; in the loin (*dans le flanc,* the region between the last rib and the crest of the ilium, on the lateral aspect of the trunk), once; in the lumbar region, once; and in the fossa supraspinata, once.

It is exceptional for the pain to radiate to the side of the abdomen corresponding to the inflamed lung, or for the pain to occur upon the opposite side of the thorax. In both instances this may lead to errors in diagnosis. [Such cases simulate appendicitis, peritonitis, gallstones, and other abdominal conditions.—Ed.]

This sensation of pain may precede the chill, therefore be the first symptom of the affection; as a rule, both symptoms occur simultaneously. With this coexistence of symptoms alone the presumption is justified that one has to do with an inflammation of the lung. If no pain has occurred at the onset of the affection, it is rare for the pain to occur during the course of the disease. Occasionally a cessation of the pain is noted after a few hours, and at other times the pain may last for days. It is very rare for the pain to assume such proportions that it is unendurable.

In all cases in which the pain is severe it shows an intense affection, because respiration is diminished, and inspiration, which is necessary to supply the required amount of oxygen, is made almost impossible.

Naturally every attempt at expiration exaggerates the cough. Sneezing also produces an increase in pain. The respiration may become very superficial and dyspnea arise as a result of the pain.

Dyspnea is a frequent accompaniment of pneumonia. The causes of this symptom are various. Relatively frequent, and especially at the onset, occasionally also in the course of the disease, dyspnea is due to the pain. Inspiration, as has already been noted, is not sufficiently deep. As a result of this, an insufficient quantity of oxygen is taken up; the blood is insufficiently decarbonized, and more or less well-marked cyanois, especially of the face, is noted. A certain proof of this causal connection is found in the fact that with the cessation of pain dyspnea and cyanosis disappear. The injection of a sufficient quantity of morphin brings this about.

Independently of the pain, marked dyspnea may occur with the onset of the chill and in the first few hours after the chill. The cause of this is difficult to determine. Very likely the dyspnea is in some way connected with the irritation of the central nervous system. It seems as if the respiratory functions had a close relation to the pathologic condition. The dyspnea, despite the further development of the disease and the occurrence of hepatization, diminishes without being especially dangerous to life. [In children very frequently, in adults occasionally, an "expiratory grunt," so-called, occurs. It is a sign of great diagnostic significance. It occurs even though pain is absent.—Ed.] Dyspnea only becomes dangerous if a diffuse catarrh is present or edema of the lungs occurs. The latter luckily is extremely rare, and scarcely ever occurs in croupous pneumonia. It is much more frequent in atypical pneumonia. Dyspnea is not dependent upon the extent of the consolidation, provided this consolidation does not affect more than one lung. Andral has already expressed this opinion. Dietl says that the patient breathes easier after hepatization has been completed than he does before. This of course does not exclude the fact that a high grade of dyspnea occurs when both lungs are affected, and at the autopsy, only a part of the right upper lobe is found to have escaped hepatization; although it may be edematous, as I saw in one case.

In cases of skoliosis, pneumonia always leads to well-marked dyspnea.

Even when there is no dyspnea a marked increase in the respiratory frequency is noted, which may last during the entire disease. In adults the number of respirations varies between 24 and 40 per minute. Such a high grade of respiratory frequency as is noted in miliary tuberculosis of the lungs, in which from 60 to 80 respirations are counted in a minute, if we except the agonal stage, is not seen in croupous pneumonia. Only in children are from 40 to 50 respirations per minute noted. [In one of our patients, a child, two and a half years old, the respirations were 120.—Ed.]

"The evening rise in the temperature is usually accompanied by an increase in the number of the respirations, which diminishes again considerably toward morning, furnishing additional proof of the fact that, while the exudate remains the same, the respiratory rate is determined chiefly by the nervous phenomena, which increase and diminish with the temperature. The dyspnea is combined with great superficiality; inspiration is sometimes effected by several short gasps, so that the speech becomes staccato (interrupted). If 'playing of the alæ nasi,' from movement of the levatores alæ nasi, occurs during the disease, it is to be regarded as a grave symptom" (Finkler, p. 35).

A further symptom which is almost invariably present is **cough.** "Cough is rarely of use; always troublesome, sometimes dangerous," v. Jürgensen justly remarks. This symptom arises with the onset of the affection. It is always noted in previously healthy patients when the lower lobes are affected; it is not marked and may be completely absent in pneumonia of the upper lobes, and in delirious patients, aged persons, and patients in whom the pneumonia is a result of other previously present diseases depending upon affection of the respiratory or circulatory organs. To this group of diseases also belong enteric fever and puerperal affections. At first the cough is noted in a few short paroxysms, during which a small amount of tenacious glairy mucus is brought up. Later paroxysms of cough of long duration may occur, which on account of their frequency may become serious, and in case they last during the night may add a great deal toward robbing the patient of his strength. Occasionally severe pains in the intercostal and abdominal muscles are noted; and once as a result of the cough I saw a rupture of the rectus abdominis. In the majority of cases the cough is endurable.

The **sputum** of pneumonic patients requires full description, at first as regards diagnosis, and then on account of prognosis. In the beginning the sputum is tough and has a glairy appearance as the result of the mucin contained in it. When the sputum is constituted in this way, there can be no doubt of its origin; it is a secretion of the bronchial mucous membrane. Soon, however, it takes on a bloody appearance; the blood is so intimately mixed with the mucus that the sputum acquires a characteristic appearance, to which the name "rusty" has been given. [The rusty sputum was noted as present in 73, or 37.4%, of 195 cases; absent in 40; and not noted in 82.—Ed.] When such a sputum is present, the diagnosis of pneumonia can be made with certainty even if but slight attention is given to other phenomena. Hemorrhagic infarct is probably the only condition that may give rise to difficulty in diagnosis. Apart from other symptoms, which will be more fully explained in the diagnosis, a differential point may here be mentioned, namely, that the sputum contains more blood and is not so tough as in pneumonia and never has such a bright red appearance. The possibility of a mistake in diagnosis rests on the fact that even in pneumonia the sputum may be much more hemorrhagic and quite uncharacteristic, even pure blood may be expectorated.

As regards the time at which the characteristic sputum occurs in pneumonia Grisolle has given exact figures. In 131 patients the sputum showed the characteristic color:

On the 1st day,	in 45 cases.	On the 6th day,	in 6 cases.
" " 2d "	" 31 "	" " 7th "	" 5 "
" " 3d "	" 14 "	" " 8th "	" 2 "
" " 4th "	" 14 "	" " 11th "	" 2 "
" " 5th "	" 11 "	" " 12th "	" 1 case.

The quantity of the sputum expectorated is astonishingly small. Grisolle's estimation places it at 64 gm. (2 ounces) in twenty-four hours. Huppert and Riesell found in a pneumonia of the entire left lung that the maximum daily amount of sputum expectorated was 67.3 gm. with 5 gm. dry substance; while the minimum amount was 35.7 gm. with 1.9 gm. dry substance. Renk found in a case of pneumonia at the middle of the attack a daily amount of sputum of 26.0 gm. with 23.66 gm. water. [Osler states that 150 to 300 c.c. (5 to 10 fluidounces) are discharged daily.—Ed.]

These facts speak also for the previously given opinion that most of the pneumonic infiltrate is not expectorated, but absorbed.

Regarding the chemical constituents of the sputum, it may be mentioned at this point that, according to the latest investigations, by Lanz, and the results of former labors, the quantity of nitrogen that combines with the sputum, in other words, the loss of albumin, is comparatively large, and is especially due to the amount of blood contained in the sputum. On account of the short duration of the disease it is, however, of slight consequence to the general organism.

Bussenius found sugar in the sputum of pneumonia of a woman who suffered from diabetes before she contracted pneumonia.

Occasionally the sputum does not show the usual characteristic rusty appearance. In some few rare cases it may be lemon-yellow, the color of brick-dust, leaf-green, and even have the appearance of prune-juice. The causes of these abnormalities have not been investigated. Only the last-mentioned consistency is of importance as regards the course of the affection, which shows an insufficient power of coagulation of the blood in the alveoli and a simultaneous lessening of the tough, tenacious secretion of the smaller bronchi, and is probably also due to a loss in power of the heart muscle. This condition, however, may be a transitory one. The prognosis, therefore, in a case in which prune-juice expectoration occurs, is not necessarily unfavorable, but must be looked upon as doubtful.

In rare cases, in the course of pneumonia, pure blood may be brought up in quite large amounts. Stricker has in his collective report of 900 cases mentioned 7 cases of hemoptysis. [It has occurred twice in our experience. A woman, aged forty, died; a lad of sixteen, in the course of a second attack which developed five days after the crisis of the first.—Ed.] In one case in which the fatal termination was due to the large amount of blood lost, the diagnosis was definitely determined by postmortem.

The great rarity of such occurrences is noted from the facts given by Stricker that these cases occurred among 16,711 cases of hemoptysis which were treated during the years from which this report was made up. I have seen but one case of very marked hemoptysis in pneumonia. [In one of our private patients the attending physician made a diagnosis of pulmonary tuberculosis because of the profuse hemorrhage.—Ed.] The affection arose during convalescence from enteric fever, and, on account of the bleeding, had a fatal termination. The autopsy showed red hepatization of the right upper lobe.

Not infrequently the sputum is altogether wanting. In children the absence of sputum is due to the fact that it is swallowed. But in pneumonia of the upper lobes other causes are responsible for the absence of sputum. Bouillaud's theory is that sputum is frequently absent in pneumonia of the upper lobe because the movements of the thorax and the paroxysms of cough by which the masses of exudate contained in the alveoli are loosened have less influence upon the upper than upon the lower lobe. I believe the diminished expectoration to be due to the same causes as the condition of the alveoli, which are less completely filled in pneumonia of the upper lobe during the stage of red hepatization: namely, to a diminished hyperemia, or, to be more exact, a diminished amount of bleeding into the alveoli of the upper lobes during the stage of inflammation. The result of this is that the accompanying secretion of the finer bronchi is less than in pneumonia of the lower lobes. More difficult to explain is the frequent absence of sputum, and especially of bloody sputum, in pneumonia of the lower lobe, which occurs in the course of delirium tremens, in insane patients, in the aged, and in the course of pneumonia after acute diseases. I can only mention the hypothesis that in such cases a stasis follows in the capillaries and veins as a result of disease of the alveolar epithelium, and produces a migration of the white blood-corpuscles in the alveoli; that is to say, that the development of gray hepatization occurs so quickly that the intermediate stage—red hepatization, due to the migration of the red corpuscles—lasts too short a time to produce bloody sputum.

In the sputum of pneumonia plugs and masses of fibrin which have been previously mentioned are found. The finding of these masses is not at all difficult if they are large and of thick caliber. It is more difficult to find the smaller ones. To do this the sputum must be spread out upon a flat plate, and especially the thick mucous plugs must be carefully picked over with a needle. Usually the coagula are found as threads rolled together; occasionally as a small coil embedded in mucus. The finest coagula appear as distinct threads, sometimes only half coagulated, which can be isolated. The sputum may also be washed out in a glass so that the threads become free of mucus and adherent cells, and in this way the coagula are more easily found (Biermer, p. 51). A very simple method has shown itself useful to me to find these fibrin coagula. Only the rusty sputum of a pneumonia patient must be placed in a large bowl with water, and a slide or a large glass plate held in the water so that such sputum without much labor may be spread upon the glass, or, more properly, spread itself upon the glass. The slide is then removed from the glass with the sputum upon it, the lower surface is dried with a cloth, and the slide is placed under the microscope without a cover-glass, and a low magnifying power is used (40 to 50). Almost exclusively the wavy fibrin threads of different sizes can in this way be seen.

The sputum also contains diplococci, as is to be supposed, from our knowledge of the pathogenesis of the affection. Wolff found in the sputum of pneumonia patients the same organisms that Weichselbaum found in the inflamed infiltrate of the lung, and especially emphasizes the diagnostic value of such a finding. He found the diplococcus in 70 cases 66 times; only thrice he found the Friedländer pneumonia bacillus instead of the diplococcus, and in only one case was neither present. The number of diplococci in the sputum varied very much, and did not allow of any deduction as to the severity of the affection. Also the time during which they might be noted varied between a few days and two weeks.

Von Weismeyer in his careful examination of the sputum in 39 pneumonias justly emphasizes the importance of determining the variety and whether different varieties of bacteria are present in the sputum. In two of his cases he was able to demonstrate in the sputum after he had carefully instructed the patient to cleanse the mouth and to expectorate into the glass after coughing, besides the Fraenkel-Weichselbaum diplococcus, cocci in chains. In 3 cases he had to do with a pure streptococcus infection. He concludes from his observation that the finding of streptococci in the sputum of a typical croupous pneumonia, without regard to the age, without regard to the strength of the patient, without regard to the fever or the local phenomena, has an important influence on the prognosis, as it is extremely likely that a very marked delay in the loosening of the infiltrate is to be expected.

Only with the help of objective methods of examination can the seat of the disease, and the changes which occur at the seat of the disease, be definitely determined.

To determine the conditions by means of **inspection** and **mensuration** is an obvious measure; it is, however, not of great value. Broussais thought that a dilatation of the diseased side occurs; Laennec and Andral saw nothing of this. Woillez in exact measurements found nothing abnormal. Wintrich, on the contrary, notes a widening of the thoracic space as the result of pneumonia if the disease occurs in the base, and is spread out anteriorly and the disease is in the stage of hepatization. The fact that this widening is better marked if the two sides are compared after complete expiration, and that the difference amounts to ½ to 2½ cm., is by no means a proof that dilatation occurs. After the explanation of the anatomic condition of the consolidated lung in pneumonia previously mentioned (p. 387), the diseased organ has at the most the volume of a normal lung distended to its utmost by forced inspiration; a dilatation of the thorax greater than the lung can, therefore, not exist. If, however, the circumference of the diseased side in expiration is greater, it is no proof of the expansion of the thorax, but that the infiltrated lung, because it cannot return to the volume of a normal lung in the stage of expiration, during its rise in expiration, prevents the corresponding side of the thorax from collapsing.

In the main, Grisolle is correct when he declares that mensuration of the thorax in pneumonia is unnecessary, worthless, and harassing.

The expansion of the thorax above the inflamed pulmonary area is not markedly diminished, unless the infiltration is very extensive (v. Ziemssen, 231). Inspiratory expansion is lessened only when the pain is very severe. The immobility of the diseased half of the thorax is, however, never as complete as in the presence of a large pleural exudate (v. Ziemssen, 235).

The most valuable results are obtained by **percussion** and **auscultation.** At the onset of the disease a hollow, slightly tympanitic note is noted upon percussion. This arises as the result of swelling of the alveolar epithelium due to the fact that the capillaries are overfilled with blood, and as a result of this the elasticity of the pulmonary structure is lessened and under percussion the pulmonary area does not vibrate as in the normal lung. The tympanitic note is the result of a loss of elasticity of the pulmonary structure due to anatomic reasons; the amount of air contained in the alveoli is no longer inclosed in so markedly stretched membranes as in the normal condition.

With the development of red hepatization the hollow note gives way to a dull note. That there is always a slight tympanitic element, as Thomas says, I have not been able to substantiate in all cases. Most frequently this tympanitic note occurs in pneumonia of the upper lobes, in which most frequently, on account of the previously mentioned anatomic reasons,—incomplete consolidation occurs, hence red hepatization is not complete,—complete dulness does not occur. Here the continuance of the tympanitic note is explained upon the same grounds as in the stage of engorgement. In those cases in which with complete hepatization of the lower lobe a tympanitic note occurs, the columns of air in the bronchi of the infiltrated pulmonary areas may be regarded as the causes of this condition, as Bäumler and v. Jürgensen declare. This can sometimes be proved by the change of the tympanitic note in opening and closing the mouth, but v. Jürgensen even admits that this is not frequent. He has seen only two cases of pneumonia of the lower lobes in which percussion developed an exquisitely tympanitic note, in which the note undoubtedly varied when opening and closing the mouth.

Finkler has called attention to a different origin of the tympanitic percussion note. If the pneumonia occurs in the left lower lobe and occupies Traube's semilunar space up to the limit of the heart-dulness, the infiltrated pneumonic area may transmit the stomach resonance to the surface of the thorax. Even percussion of the heart may, under such circumstances, give rise to a tympanitic note.

The area of dulness usually coincides in shape with the affected pulmonary lobe, but its extent somewhat exceeds the limits of the normal lobe. Dulness noticeable only over the back, unless confined to the fossa supraspinata, is to be referred to the lower lobe. Dulness occurring only on the anterior half of the chest belongs to disease of the upper lobe; one noted upon the right, in the axillary space, to disease of the middle lobe. Although the lower lobe upon both sides only reaches to the middle of the shoulder-blade, in the stage of hepatization it takes in nearly the entire posterior surface of the affected side, whereas the upper lobe, when it is alone affected, spreads laterally and posteriorly. Although statistics teach us that the lower lobes, especially the right, are more frequently affected, it is nevertheless entirely unjustifiable and frequently of decided detriment to the patient to examine only the posterior surface. In this respect not only the anterior surface of the chest, but the axillary region as well, should be examined at every visit, as many pneumonias, both of the upper and of the lower lobe, first show hepatization in the axillary region, and many pneumonias which begin centrally first reach the surface in front or at the sides, and in this way become accessible to the methods of physical examination (Gerhardt, p. 260).

Von Jürgensen believes that pneumonia of the lower lobe as a rule becomes noticeable first in the posterior axillary line near the lower border of the scapula. In children he percusses first anteriorly and then posteriorly in a girdle-like manner. The child lies upon the lap of the mother or sits up; the arm of the side that is to be examined is slightly removed from the thorax, while the head is to be supported with special care.

As regards the size of the pulmonary area which may be determined by percussion as dull, Wintrich says: "Pulmonary tissue devoid of air and peripherally infiltrated, gives a short and weak note only if the space devoid of air has a diameter of about 5 cm. and a depth of 2 cm., and then only upon superficial percussion."

The tissues surrounding the area of consolidation also show changes in respect of percussion. If the parts of the lobe contiguous to the thoracic wall are in the primary stages of the affection, whereas the central part is already advanced,—a condition which belongs to central pneumonia,—the percussion note elicited over this lobe, may be conspicuously full and deep, compared with which the note over the normal side appears high in pitch and abnormal (Skoda's resonance). It occasionally happens that at the onset of the affection a mistake in diagnosis is made as to which is the affected side, which, however, in the later stages of the disease can readily be cleared up. Rarely under such circumstances may it occur—it happened to me but once—that the pneumonia upon the second day shows a critical defervescence, and it is then impossible to determine accurately which side was the diseased one.

Further, it may occur that where one lobe is diseased the neighboring healthy lobe may give rise to a *tympanitic note* upon percussion. This occurs most frequently over the right apex, if the middle and lower lobes are the seat of the inflammation. The reason for this tympanitic note, then, is the increase in size of the infiltrated lobe up to the volume which it would acquire under normal circumstances at the height of inspiration. From this a decrease in space of the sound upper lobe results, and a lessening of the expansile power of its elastic tissue. This tension, however, in the normal thorax gives rise to the full deep note upon percussion.

Finally, there must still be mentioned that over the neighboring sound lobe a *cracked-pot sound* or, to speak more briefly, ringing may be elicited upon percussion. I explain the origin of this sound by the unequal effect of percussion at the border between consolidated and completely empty pulmonary tissue. The origin would, then, be analogous to the phenomena of interference in tones. My reasons for this conception are found in the observation of a similar note over a dilated and hypertrophied stomach-wall. The advance of the contraction wave is occasionally visible through the abdominal wall, and if percussion is practised at the junction between such a wave, produced by contraction of the muscles and the neighboring relaxed part, a cracked-pot sound is obtained. Therefore, also in the lungs the irregular tension of the tissue at the border between the diseased lobe and the sound structure may be looked upon as the reason for this phenomenon, and the air contained in the bronchi may be excluded as a cause of this ringing sound.

Just as many-sided and important are the results of **auscultation.** Stokes believed that the first stage of pneumonia might be diagnosticated from the sudden appearance of a local *puerile respiratory murmur,* combined with fever and excitement on the part of the respiratory system. He frequently found, in cases in which the inflammation spread toward the apex, that puerile respiration preceded crepitant râles by some hours. That this was no general, but only a partial circumstance was shown in the fact that this puerile respiration was much more pronounced in the neighborhood of the seat of the affection than in the opposite lung.

Such an increase in the inspiratory sound is easily understood *à priori,* as the puerile breathing, in the first place, is dependent upon the difference between the alveolar space and the lumen of the bronchiole which empties into it, so the hyperemia of the pulmonary tissue which occurs at the onset of pneumonia may very easily be the reason of the narrowing of the bronchus which ends in the affected area. According to Rossignol, it must be taken into consideration in children that the width of the bronchiole terminating in the infundibulum is as 1 : 3, while in adults it is as 1 : 2.

The statement of Stokes can nevertheless not be looked upon as authoritative, as this condition is not proved. As it is often impossible in the first few hours to examine a case of pneumonia, a general corroboration would be very difficult to obtain.

If, on the other hand, Grisolle gives vent to the opinion that an enfeeblement of the respiratory murmur at the onset of the affection exists, there may perhaps be no contradiction between the two statements; both conditions may occur. But even the latter symptom has not especially been noted by any one else. [The editor agrees with Osler in supporting Grisolle in this respect.]

The **crepitant râle** remains the characteristic sign of the inflammatory engorgement of the pulmonary tissue, as Laennec says. It may be noted from the first moment of the inflammation, and gives an impression of the bursting of very small, uniform, slightly moist bubbles. The respiratory murmur simultaneously may be very plainly heard. This important sign of beginning pulmonary inflammation is always present, and is found in only two other conditions—edema of the lungs and hemorrhagic infarct; two affections which, in general with the symptoms belonging to them, may be easily differentiated. It appears from these statements that Laennec could not have considered the crepitant râle a specific symptom of croupous pneumonia—as is the view of some of our later authors. It occurs in all conditions in which disease originates in the vesicular structure of the lung, and the finest bronchioles become involved before the alveoli are airless and filled with solid exudate. The crepitant râle can only be explained in the manner that in the finest bronchioles, or, more properly, at the point at which the bronchiole empties into the alveolus, a tough, previously present fluid is broken by the inspired air, which still, however, with difficulty is able to reach the vesicular structure of the lung. For this reason this râle may also be heard in other diseases besides those described by Laennec: in fever, at the bases posteriorly, when patients are unable to change their position (Walche, Barth and Roger, Wintrich, v. Jürgensen); in the primary stage of acute catarrh of the finest bronchi (v. Jürgensen). I have repeatedly in cases of tuberculosis heard distinct crepitation over the upper lobe, not only transitorily or occasionally, but for quite a long time.

In respect to the origin of the crepitant râle, it can be easily understood, when in the place of this physical sign occasionally fine moist râles are heard. This occurs in connection with the spread of the catarrh of the bronchi, especially of those of larger lumen. This explains the fact that Hourmann and Déchambre have in the aged occasionally not heard this characteristic crepitation, but large moist râles, and Bouchut has noted an analogous condition in children up to the third year of life.

Later in the course of the affection, in some cases, a *prolongation of the respiratory murmur* occurs. As the origin of this may be derived from analogous conditions, as bronchial breathing, it may be assumed that the prolongation of the expiratory murmur may be due to the almost complete absence of air in the alveoli, but the dislodgment of air has not as yet been complete. [After a cough harsh or broncho-vesicular breathing may be heard at this stage.—Ed.]

Well-developed *bronchial breathing*, heard in inspiration as well as in expiration, occurs in the stage of red hepatization. In typical cases this is very characteristic. One has the sensation that it is very close to the ear and is not transmitted by any substance lying between. But not rarely it is absent or it is not as characteristic, as has just been stated; this is particularly the case when it is most important for diagnostic purposes. Absence of bronchial breathing is particularly frequent in pneumonia of the upper lobes, or of the anterior surface of the chest. Its absence has to do with incomplete red hepatization; the alveoli are not completely empty of air, as not enough blood has entered into them. It is not possible for quiescent columns of air to be produced in the bronchi that supply the diseased upper lobe. As I have proved in another place,[5] bronchial breathing arises in the diseased lung only from the fact that the column of air drawn into the sound parts of the lung, or expired from these parts, passes the column of air leading from the bronchi of the diseased lobes. As long as there may still be air drawn in to the partly filled alveoli of the upper lobe the production of quiescent columns of air in its bronchi is not possible; therefore the conditions necessary for the production of bronchial breathing are not at hand.

I believe that I may mention at this place that it occasionally is no easy matter, after a physical examination, to say whether dulness of the entire upper lobe is of a tubercular or pneumonic nature, and then, if the microscopic examination shows nothing definite, several days may elapse before a diagnosis may be made with certainty.

It may also be very difficult to differentiate the symptoms if to a pneumonia of a left lower lobe a pleurisy and a pneumonia of an upper lobe of the same side are added. Some time ago I noted just such a case. Anteriorly upon the left, reaching from above downward, complete dulness, everywhere complete suppression of breathing, decreased pectoral fremitus, displacement of the heart, and no alteration in the position of the spleen were noted. After the purulent effusion was removed by means of resection of the ribs, dulness still remained upon percussion over the upper lobe; that is, the pneumonia remained behind, but later disappeared completely. The patient was discharged cured.

Bronchial breathing may be absent if the bronchi which lead to the part contain a great deal of mucus. Very likely it is always due to the fact that a large number of the smaller bronchi are occluded. I think it exceedingly unlikely that in the case of pneumonia the larger bronchi can be completely filled with mucus or other material.

An important symptom is *bronchophony;* that is, the auscultation of the vibrations of the vocal cords in speaking, counting, etc., by means of their disseminated waves of sound. If one auscultates over a pneumonic infiltrated lobe while the patient speaks, one hears the spoken words very plainly. It appears as if the waves of sound immediately enter through the thoracic wall into the ear of the person auscultating, "but sounds in bronchophony are never as plain and distinctly articulated as if the words came directly from the mouth of the one under examination" (Wintrich, p. 147). This sound production, which cannot be heard over the normal lung, Laennec explains from the property of conduction of the consolidated pulmonary tissue which is better than in the normal condition; whereas Skoda believes the same to be due to the consonance of the air in the bronchi of the consolidated pulmonary areas, which are easily capable of transmitting the consonant voice. A powerful, high-pitched bronchophony is especially characteristic of pneumonia.

According to my opinion, the causes for the production of bronchophony are the same as those which give rise to bronchial breathing: the presence of quiescent columns of air, therefore the absence of the expiratory air-current, and the airless condition of the alveoli. Only in quiescent columns of air is the transmission of the vibration of the vocal cords into the pulmonary tissue possible. The fact that along the trachea, in spite of a constant movement of the column of air, bronchial breathing and bronchophony may be heard, does not stand in the way of this deduction. The transmission is made possible here that the sounds are transmitted from the vocal cords a certain distance by means of the resisting cartilaginous wall of the trachea and main bronchi.

During bronchophony, in pneumonia, one hears at the end of a spoken word, occasionally even at the end of each spoken syllable, lasting somewhat longer than each separate syllable, a rough, short puff. This is nothing more than the expiratory bronchial respiration, heard between the pauses of the vibrations of the voice, and due very likely to the more passive expiratory collapse of the thoracic wall.

Rarely in the course of pneumonia may *egophony* be noticed. According to Laennec, egophony is the result of the natural resonance of the voice in the ramifications of the bronchi, transmitted by a thin and tremulous layer of fluid and later becoming more distinct on account of the compression of the pulmonary tissue, which allows the latter to become denser than normal, and therefore a better sound conductor.

Skoda entirely disagrees with this conception, with very exact proofs. Here only the fact may be emphasized that egophony may occur without pleural effusion, although it occurs most frequently under those circumstances. Skoda thinks it extremely likely "that Laennec's egophony is produced through one solid body striking against another solid, liquid, or gaseous body. These shocks, however, cannot take place inside the thorax, unless the voice vibrates in unison with them in a space filled with air," because the pulmonary tissue is not set into vibration from the larynx through the walls of the trachea and the bronchi. It is, therefore, likely that in most cases the walls of the bronchus, in the interior of which air is vibrating, react by impinging upon the contained air, and thus produce the quavering sound. It is, however, also possible that occasionally a portion of mucus, which incompletely closes the opening of a bronchus, may imitate the mouthpiece of a whistle and gives rise to the quavering sound.

Wintrich is opposed to this conception: "The sounds of the voice become bleating under the same circumstances under which the nasal sounds are produced, only with the difference that, in the case of bleating, the transmission of the spoken sounds is rapidly interrupted in a tremulous manner. Egophony appears, as far as the timbre is concerned, to be nothing more than an exaggerated bronchophony."

I conceive egophony to be an expiratory staccato interruption of the bronchophony over the consolidated portions of the lung. This staccato interruption is due to the fact that the expiratory diminution of the thoracic space is not produced by a gradual, steady retraction of the thoracic wall, but is effected in an uneven, jerky manner. Egophony heard over consolidated portions of the lungs is therefore analogous to the speech of some healthy persons who can only speak in a trembling voice.

The auscultatory phenomena described up to this time, bronchial breathing and bronchophony, are especially prominent in the stage of red and gray hepatization. Râles are very much rarer in these stages. They are only to be looked upon as signs that the accompanying bronchial catarrh, which is always present in a greater or less degree during a pneumonic infiltration, becomes somewhat more intense, and that secretion is plentiful in the bronchial tubes. This occurs most frequently in the stage of transition from gray hepatization to purulent resolution. Characteristic of purulent resolution, however, is crepitation, the *crepitatio redux*. It is a sign that the alveoli have become free of exudate, and that air is beginning to enter into them. The physical conditions are exactly the same as at the onset of the affection, the entering air must break up the narrow opening of the bronchiolus into the infundibulum.

Next to percussion and auscultation, **palpation** must be utilized in the diagnosis of the affection. This method is of especial value in testing the *vocal fremitus* of the consolidated pulmonary parts. It is for the most part increased, and then, in connection with the other characteristic symptoms, a proof of pneumonic consolidation. It also enables one to exclude a pleural effusion from pneumonia. Its absence, however, does not allow of such a deduction. Like bronchial breathing it may be temporarily absent, especially if there be a plentiful secretion in the bronchi which lead to the part (Skoda). A single paroxysm of cough often suffices to make the symptom reappear; or it may be permanently absent; even a diminution of vocal fremitus, instead of an increase, may occur. In cases characterized by abundant hepatization, in which, at the autopsy, deep rib-furrows are found on the surface of the lungs, the increase in the vocal fremitus is often missed not only temporarily, but during the entire course of the disease. The powerful unilateral pressure on the thoracic wall prevents the latter from vibrating (Gerhardt [84], p. 259).

On the whole, it may be taken for granted that an increase in vocal fremitus in the majority of cases of croupous pneumonia is present, or at least may be noted upon frequent examination. It must, however, be mentioned that such a careful observer as Grisolle has frequently found the fremitus diminished rather than increased.

To sharply define the neighboring borders in which in one part the fremitus is present or even increased, whereas in the other the fremitus is weakened or entirely absent, Wintrich advises not to coapt the entire hand, but only the narrow, inner border of the same. The examination will be more exact if mediate palpation is used and the fremitus transmitted to the hand by a medium a line wide, such as the handle of the percussion hammer or the edge of a shaving. Wood transmits the vibrations of the voice splendidly.

Worth mentioning is the fact, determined by Graves, that by means of palpation a "synchronous beating of the heart's action may be noted in a marked dissemination in the first stage of pneumonia." It is easy to understand, says this author, how in a soft, overfilled, and half fluid condition the lung, which is connected with the heart by such large vessels, may pulsate with such force as to be similar to the condition occurring in aneurysm.

In connection with this it must be mentioned that, according to Skoda, the auscultator notes a shock over the hepatized area not infrequently with every pulsation of the heart. This does not arise, as Laennec supposed, through a transmission of the heart-beat, but through the pulsation of the arteries in the hepatized lung.

Palpation as a means to determine the degree of heat of the normal and diseased organs contained in the cavities of the body, in short, **thermo-palpation,** has been recommended by Benczúr and Jónas. They found that in stroking the skin with the flat of the hand differences in temperature of the surface, corresponding to the organs contained in the cavity of the body, can be determined with certainty. The skin over organs containing air always feels warmer than the skin over organs devoid of air, so that in this manner the boundary between air-containing organs and solid organs can be determined as certainly as by means of percussion. Accordingly one also finds, under pathologic circumstances,—for example, in pneumonia,—a temperature deviating from the normal. In special cases in which, for example, a rapid determination is necessary, or in which percussion cannot be used, as in case of fracture of the rib, hemoptysis, or aneurysm, thermo-palpation may be exceedingly useful.

That in these cases there actually were differences in temperature was determined with the thermogalvanometer, with differential air-thermometers, and with mercury thermometers especially constructed for this purpose.

In a later communication these authors explain the reasons for the results of thermo-palpation in the individual parts of the body. They found that the thermo-palpatory boundaries were not determinable over carefully covered parts of the body, as also in a warm bath, under water, and were noted only when the covering was taken off or when the individual on leaving the warm bath was treated with a cold affusion. This condition of the thermo-palpatory boundaries obviously indicates either that the two parts of the body on either side of the thermo-palpatory border-line do not cool off evenly, or that after a simultaneous cooling the warming which follows takes place with a different rapidity. The absolute temperatures of the respective internal organs are not the main conditions, but the quantity of blood which in a given time is brought in contact with the surface of the body by these organs.

On the normal thorax there is a fairly constant relation between the results of percussion and the temperature differences observed on thermo-palpation, areas that are dull on percussion corresponding to areas that appear colder on thermopalpation; thus, for example, the skin is always cooler over the heart and liver than over the neighboring healthy lungs. On the abdomen there is not such constancy between the topographic divisions of the temperature differences and the results of percussion. This phenomenon finds its explanation in the inconstant conditions of the circulation of the abdominal cavity. The stomach and the bowels,—depending on the degree of fulness or emptiness, on the stage of digestion, and on the quantity and tension of gas contained in those viscera,—may, in thermo-palpation, be warmer or colder, in comparison to one another and to the liver, according to the rapidity of the circulation in their walls, which is greater, for example, during digestion, and less in meteorism. Just so, under pathologic conditions, a tumor rich in blood-vessels may at one time be cooler and at another time warmer than the contiguous bowel.

Under favorable circumstances one may even be able to feel differences in warmth between two organs that give a dull note upon percussion, or between two pathologic products; for example, the pneumonic infiltrated lung and the liver; pneumonic lung and spleen; spleen or liver and ascites. In this way one will be able to determine boundaries which cannot be noted upon percussion.

In corroboration of the observations of Benczúr and Jónas, Fritz in especial arrives at the result that by means of thermo-palpation the primary stages of phthisis may be proved with certainty; that in the case of pleurisy the first, that is, the inflammatory stage which precedes exudation, cannot be determined by thermo-palpation; that in pneumonia, as Anrep has already proved, the recently inflamed lung is warmer than the normal, and much warmer than that part which has entered into the stage of gray hepatization.

Meissner, on the contrary, could not demonstrate to himself through simple palpation the thermic differences between organs containing air and solid tissue; a thermo-electric test, however, gave him the exact opposite of the conditions which had been communicated by Benczúr and Jónas. He found without exception that the skin surface over the heart (absolute heart dulness) was warmer than that over the lung.

These conspicuous differences between the results obtained upon examination by means of thermo-electricity and of manual examination require further investigation. So far as the latter are concerned, my own observations do not permit of any doubt as to the correctness of the observations of Benczúr and Jónas. I have frequently enough made control experiments, and from the first day of the affection I have been able to establish the certainty of the results. The value of the results obtained by thermo-palpation, however, is another question. Up to the present I have not been able to learn any more than by the other methods of physical examination.

Finally, there must still be mentioned that Wintrich has called attention to a combination of palpation and percussion. This takes into consideration, besides the determination of the acoustic phenomena, the accompanying sense of resistance according to the character of the organs under examination. Von Jürgensen advises practising this method to educate the sense of touch, which is usually greatly neglected, in addition to the sense of hearing. Especially in the pneumonia of children this method of examination is of the greatest value. [To determine the sense of the resistance is most valuable and often convincing when the character of sounds is doubtful.—Ed.]

Palpatory percussion shows a distinct increase in resistance in all those parts in which solid tissue is contiguous to the thorax-wall. An increased sense of resistance occurs in pneumonia, in pleurisy with large effusion, and in diffuse total carcinomatous degeneration of the lung. Without doubt this increase in resistance has to do with the diminution in the lumen of the smaller bronchi, which in the latter case is the most complete. The lumen of the bronchi—that is, the air contained in them—represents the coefficient of elasticity for the consolidated tissue, and is inversely proportionate to its resistance.

A special description is required as to the behavior of the sound, or, more correctly for many cases, the apparently sound side. Even Laennec had noted that the pain may be referred to the other side of the lung, without inflammation having taken place upon that side. Gerhardt gives as an explanation for this fact, that the anastomoses between the intercostal nerves on both sides in the anterior mediastinum made it possible for the sensation of pain to be transmitted. Huss in one out of eight cases in which he made a postmortem examination demonstrated in the fourth anterior intercostal nerve upon the right side a branch which, after supplying the triangularis sterni muscle of the left side, communicated, by means of a very fine branch near the left sternal border, with the corresponding nerve of the left side.

From this, however, only the occurrence of pain in the neighborhood of the sternum is explained, but not the pain which is more noted laterally or in the region of the shoulder, which occurs upon the side not affected by the pneumonia. Such cases I have also a few times had an opportunity to see. According to my opinion, the pleura of the apparently sound side was also in a slight degree affected. The local inflammatory changes may even go back further than this. I make this assertion from an observation in which, only after the crisis, a pleural effusion occurred upon the side which up to that time had remained healthy.

Transmission of bronchial respiration and of râles from the affected side to the sound side I have never noted. [In consolidation at the root of the lung we may have transmission of sounds to the healthy side.—Ed.] But once I noted the occurrence of large mucous râles in the left lower lobe, whereas the pneumonia affected the right lower lobe. In this case the affection lasted several weeks, with continuous fever, on account of a right-sided pleurisy which accompanied the pneumonia, and which, in spite of aspiration, only disappeared later on spontaneously. The patient, who was a physician, was tapped three times at his own request, a quarter of a liter of serous fluid being removed at each tapping. The râles upon the apparently healthy side were constantly present, and for at least three weeks were plainly audible. After return to health, they completely disappeared.

The occurrence of such râles shows conclusively that one had to do with only an apparently healthy side. Most likely the pneumonic process in such cases, as also when there is a stitch in the side upon the healthy side, fails to attain its full development.

The variations in the body-temperature and the changes in the pulmonary tissue, described so far, are associated with a series of changes, chiefly functional in character, in some of the vital organs. The explanation of the intermediate causes of these functional changes has given rise to many views. Especially v. Jürgensen was of the opinion that the fever was the special cause of these. This is, however, decidedly contradicted by the circumstance that there is no proportion between the height of the fever and the severity of the affection in the other organs. The most likely cause will be found in the fact that changes in the blood produced by the metabolic products of the bacteria are the cause of these disturbances. [The infection of other structures or the occurrence of toxemia gives rise to the following symptoms.—Ed.]

Based upon this view, a distinct line may be drawn between symptomatic involvement of other organs and pathologic changes of these organs occurring as complications; that is, through the supposition that in this symptomatic involvement only those manifestations which are due to the products of metabolism of the bacteria appear, whereas in the case of complications in other organs the Diplococcus pneumoniæ itself takes root in these organs.

The circulatory system is chiefly involved. The pulse is nearly always increased in frequency. In connection with the dyspnea mentioned above at the onset of the affection, the pulse may become very frequent, while in the further course of the affection it may become very much slower. This frequency in the onset of the affection is not so unfavorable as if it should develop in the further course of the affection.

The highest pulse-rates occur in childhood; "the younger the child, the more frequent the pulse at the same temperature" (v. Ziemssen[265], p. 217).

In adults the pulse frequency reaches 100 per minute and over. Griesinger gave a prognostic importance to the frequency of the pulse. In patients in whom more than 120 per minute occurred, more than one-third died. Opposed to this it must be especially emphasized that the pulse frequency in itself does not admit of any prognostic estimation. In the absence of delirium, and if there be not too great an increase in the respiratory rate, an increased heart's action is in itself not an unfavorable sign.

The ratio between the respiration and the pulse in croupous pneumonia is always different from what occurs under normal circumstances. In healthy individuals the ratio is as 2 : 9. To each respiration there are 4.5 beats of the pulse. In croupous pneumonia there is always such a shifting that the respirations are relatively more frequent than the pulse. This condition is recognizable at a time when the physical examination may still be entirely negative, and lasts longer than the disappearance of the fever. Prognostically as well as diagnostically this sign is of great importance. In severe cases the frequency of the respiration approaches the frequency of the pulse. If there is a high grade of atheromatous degeneration in the arteries, the respiratory frequency may be greater than the frequency of the pulse (v. Jürgensen[117], p. 68). According to my investigations, the pulse frequency as well as the respiratory frequency never reaches such an enormous height as in the case of acute miliary tuberculosis of the lungs. In excessive frequency of the pulse this fact requires notice.

Irregularity of the pulse, which in previously existing myocarditis is handed over as a symptom in this affection, may also occur in a heart previously healthy. Should this occur at the acme of the disease, which fortunately is rare, the outcome will be questionable. On the other hand, an irregularity after the crisis is more frequent, and is without unfavorable prognostic import. [In the aged, as also in right-heart dilatation, the pulse may be small and feeble. —Ed.]

The pulse-rate, after the crisis, frequently falls from 40 to 60. "It also happens that the pulse becomes slower before the onset of the crisis, so that the occurrence of the crisis may be predicted" (Finkler, p. 56).

Just so, even if rarely, the pulse may become dicrotic in the course of the disease as well as after the crisis. [On the other hand, it may be small, soft, compressible, and rapid, and the heart weak, due to toxemia affecting the vasomotor centers.—Ed.]

Especially worth mentioning is the gallop rhythm of the heart to which Fräntzel called attention in inflammation of the lung (three heart sounds during one cardiac cycle, heard evenly distributed over the entire precordia, two of them during diastole). In fifteen patients this gallop-rhythm was especially marked; twice it occurred with other serious symptoms—especially great nervous unrest—which immediately preceded the crisis; four times it occurred upon the day following the crisis with symptoms of general collapse. In all of the other cases it was either the first symptom or an accompanying symptom of cardiac weakness occurring at the acme of the affection. Twice there could be very distinctly heard how gradually the gallop-rhythm, and with it the diastolic second sound, disappeared; how the force of the heart muscle rapidly became weaker; and, after a few hours, in spite of all cardiac stimulants, death occurred. [Embryocardia is recognized.—Ed.] Among these last-mentioned nine cases it was possible to retain life only once, after the gallop-rhythm showed itself as the dangerous symptom of cardiac weakness for two days.

Arterial tension presents wide variations, as in pneumonia there is no characteristic pulse; the pulse usually shows the characters common to fever. Sphygmographic tracings show that, as a result of the lessened tension, the dicrotic wave becomes more distinct, whereas the tidal wave almost completely disappears. The apex of the curve becomes high, with steep elevation and steep decline. After the crisis, however, the apex of the curve becomes extraördinarily low; the tidal waves are numerous, and occasionally quite as high as the pulse-wave itself, whereas the dicrotic wave becomes very slight, so that the tidal wave can scarcely be distinguished from the dicrotic wave (Finkler, p. 52).

The heart itself frequently shows essential changes; its volume is enlarged, according to Corvisart; this is constantly the case if death occurs. Piorry explains this enlargement of the heart from the difficulty of respiration. Heitler is of the opinion that it is due to a texture affection of the heart. My view of the different functional disturbances which have been previously mentioned makes me explain the dilatation as a weakness of the cardiac muscle, which is a consequence of the deleterious bacterial products brought to it from the blood.

Independent of this enlargement of the heart, or accompanying it, changes of the heart occasionally occur. I have seen the heart forced over 3 cm. to the right of the sternum in the case just mentioned in which pneumonia of the left lower lobe was succeeded by one of the upper lobe, and a purulent pleurisy followed. That the pneumonic infiltrated upper lobe was the principal cause of the displacement was obvious from the fact that after the pus was removed by resection of the ribs, the heart remained in its abnormal position, and only after complete recovery of the apex pneumonia it regained its normal position.

A soft systolic murmur over the heart is occasionally heard in pneumonia, as it frequently is in weakness of the heart from other causes. Special or dangerous symptoms are not combined with it.

Pässler and Romberg's experiments, which, they claim, show that Pneumococcus, Bacillus pyocyaneus, and Bacillus diphtheriæ, acting on the center in the medulla, produce the symptoms usually ascribed to cardiac weakness by paralyzing the vasomotor nerves, are not applicable to man. For it is distinctly stated, at least with reference to Bacillus pyocyaneus and Bacillus diphtheriæ, that the experiment was followed by slowing of the heart action, whereas in man, when the gravity of the pneumonic process is enhanced by a cardiac complication, the latter is invariably characterized by acceleration of the heart action.

The occurrence of collapse at the onset, during the course, and with the termination of pneumonia is dependent on a more or less high grade of asthenia of the cardiac muscle—more frequently than on the above-mentioned embolic processes. At the same time, I think it very likely that the nutritional disturbance of the heart caused by the bacterial products of metabolism [the toxemia] more completely explains the occurrence of collapse than the view of v. Jürgensen. According to him, in a part of the cases, the increase of temperature which suddenly occurs in connection with increased resistance to the circulation (at the periphery, during the chill; in the lungs during the inflammatory engorgement) causes the weakness of the heart; in other cases, however, the lessening of the temperature produces the collapse by lessening the temperature of the trunk. In the latter cases he explains the lessening of the temperature—according to the view of Liebermeister, that an increase in the temperature acts as an irritant upon the heart—from the circumstance that in a sudden diminution of this anomalous fever irritation the anatomically influenced heart during the course of the fever cannot suddenly dispense with this irritation and reaches a lessened activity in keeping with the outcome of this irritation which shows itself as collapse. According to this, at one time an increase in temperature, at another time a decrease, would cause analogous results.

From the instructive explanation of Wunderlich in reference to collapse in febrile affections the deduction may be drawn that in pneumonia collapse, which rarely occurs with a high, even a very high, temperature of the trunk, must be differentiated from the collapse of the critical defervescence. In the first case, despite the high temperature of the trunk, the tip of the nose or a greater part of the face, in fact, the whole face, and the hands are cold and limp. The color of the face is at one time pale, somewhat yellowish; at other times a pale, livid red. The expression is more or less distorted, the turgescence at least partly lessened; the pulse is never slowed; on the contrary, it becomes accelerated, small, thready, and irregular during the collapse. The respirations in many cases become more difficult, and cyanosis may result. The functions of the nervous system are not rarely changed during collapse; haziness, confusion, stupor, unrest, trembling, convulsions, or profound prostration show themselves frequently.

In cases of this kind, which were observed by me, the pulse was always increased in frequency and small in volume. Perspiration did not occur. The whole condition gave an appearance of great gravity. If the attack was severe, it began gradually and lasted from six to twelve hours. In milder cases it occurred suddenly and disappeared rapidly. Luckily such a collapse is rare. According to my estimate, it shows itself in about 0.5 per cent. of all cases of pneumonia. A serious outcome is usually prevented by taking the proper precautions.

The second form of collapse described by Wunderlich in pneumonia, the collapse occurring during the definite defervescence, shows itself in two different ways: either it indicates the onset of defervescence, and this is more rarely the case, or it occurs after defervescence has already begun. The first may occur during the night, often during sleep, or now and again may occur during daytime. Sensations of cold, cold perspiration, difficulty of breathing, with confusion of ideas and incoherent speech, show themselves during the fall of the temperature. As a rule, this collapse also passes over without danger to life; but it may last for several days, with increase of the dangerous symptoms which remind one of the algid stage of cholera. Here, also, recovery may occur with decrease in the respiratory frequency, slowing of the pulse, which eventually may go down to 40 and lower, with the onset of a quiet sleep.

The second form of defervescence collapse is more frequent. The defervescence has begun in a normal way. All symptoms of the fever have begun to disappear rapidly; and convalescence has set in. The temperature, however, does not remain normal, but becomes subnormal. The patient has conspicuously cold hands and a cold nose, the face has a sunken appearance, and he complains of uncomfortable sensations. Usually this condition easily passes over, and only under very unfavorable circumstances, or if very improper measures are taken, becomes serious. Usually there is an intense laboring, even with a rapidly lethal outcome, during the time in which defervescence is far advanced in complications in marked alcoholic cases. Here it may occur that suddenly and unexpectedly a collapse which rapidly increases, and which brings the greatest danger even after all symptoms have been most favorable for some hours and for some days, may unexpectedly terminate in death. In aged and decrepit individuals, especially if any further unfavorable occurrence takes place (for example, a sudden profuse diarrhea), after an advanced defervescence a collapse of a very high grade may also occur, last for some time, and occasionally even then, after several days, give rise to a fatal issue.

According to my observations, also, it will be worth while to differentiate the collapse which occurs at the onset of pneumonia, during the course of the affection, during the stage of defervescence, and, fourthly, that following the crisis. In the sequence just mentioned the severity and gravity of the condition increases. I myself have luckily seen but two patients who, several hours after the crisis, after suddenly sitting up in bed, have fallen over and almost momentarily, it seemed, died—a positively agitating event at a time when all danger is believed to be over.

The **blood** itself also shows important changes which have especially been studied lately. Böckmann has determined that in pneumonia, as in other acute febrile diseases, the number of erythrocytes is inversely proportionate to the degree of temperature, while the number of white blood-cells is directly proportionate to the same; that is, that between these two structural elements of the blood during the fever there is a certain antagonism, in so far that in an increase of temperature a diminution of the red blood-cells always goes hand in hand with an increase in the white blood-cells.

A considerable number of investigations are added to this communication, of which, in the works of Rieder and Tuman, particular reports are contained. Here will only be mentioned that v. Jaksch confirmed the reports of Tuman in reference to leucocytosis in the pneumonias of childhood. According to his observations, the proportion between the white and red blood-cells ranged from 1 : 40 and 1 : 60 to 70. This symptom disappeared with the pneumonia. Occasionally this change in the blood may be a valuable help in diagnosis.

[The leucocytes were carefully studied by us in 48 cases. In 7 the toxemia was marked; the average leucocyte count was 25,900. Six of the seven died. One of the cases terminated in abscess, in which case the leucocytes were 43,800 after the crisis. In 5 cases the toxemia was medium. The average leucocyte count was 17,320. Four cases died. In 36 cases the toxemia was mild. The average leucocyte count was 23,278. Three of the patients died.

Generally the leucocyte count was greatest just before the crisis. The count showed a steady increase up to the time of crisis, and with or after the crisis a more or less sudden drop. It did not fall with the pseudocrisis, and hence continued leucocytosis may indicate the presence of pseudocrisis and the absence of true crisis. The highest count was 49,200; result, death. The highest of cases that recovered was 45,200.

In one of my cases the crisis took place on the seventh day, when the leucocytes were 43,000. On the tenth day they were 15,800. In another the leucocyte count was as follows:

14,600	on fourth	day.	
17,000	" eighth	"	
18,600	" ninth	'	Pseudocrisis.
18,000	" tenth	"	Crisis
6,450	" eleventh	"	

—Ed.]

Lähr as well as his predecessors states that the height of the temperature and the leucocytosis go hand in hand, but he could not confirm the assumption of Böckmann that this was always the case. He further says that before the crisis of pneumonia leucocytosis decreases, and after this has been complete, the blood returns to its normal condition. If, however, the number of white blood-corpuscles upon the next day again rises several thousand, one must be prepared, in spite of the continuance of the afebrile temperature, to look for a new fever outbreak or some complication. [Continuous absence of hyperleucocytosis, a leucopenia, indicates gravity, as it is only likely to occur in malignant cases.—Ed.]

Next to the heart the **nervous system** is to a marked degree concerned in the symptomatology of pulmonary inflammation. To the most frequent phenomena belongs the cephalalgia. Louis reports that in 57 cases it was absent but 8 times. However, this pain is rarely so great that the patients complain of it. It may, however, become unbearable during the paroxysms of cough. The patients then fear every attack of cough, endeavor in every way to suppress it, and during the coughing attack press their temples with their hands because this produces some diminution in the pain.

Insomnia or brief, restless sleep is not more frequent than in other febrile affections, provided the patient is not disturbed by coughing.

This cough, however, in itself alone is a frequent cause of loss of sleep, in spite of decided inclination on the part of the patient.

The opposite, conspicuous *somnolence,* is always a very dangerous symptom, and rarely occurs in cases that terminate in recovery (Wunderlich, III, p. 476). I have observed the condition several times in children, but without the ominous significance mentioned.

Frequently in children pneumonia shows its onset with *convulsions.* In the further course of the affection this symptom rarely arises again. Only in special cases, according to v. Ziemssen's communications, do convulsions occur in children—partly general, partly local—from the onset to the end of the attack.

Just so, conditions of *coma* which may have a longer or shorter duration occur, especially in children.

Rilliet and Barthez do not think it proper to class pneumonias to which brain symptoms are added as a special variety. I entirely coincide with this opinion. Even if lately Waldheim, in connection with v. Ziemssen and Thomas, establishes a special cerebral form, I do not think it justified, either by the questionable fact that in the beginning or during the entire course of the affection pneumonia may be overlooked, on account of the cerebral symptoms, or by the questionable assumption that through these symptoms on the part of the nervous system a greater danger to life is produced.

Those cases of pneumonia of children in which at the onset or in the course of the affection, with and without convulsions or vomiting, a conspicuous stiffness of the muscles of the neck occurs deserve special mention. Weber has noticed such contractions in the pneumonia of children. The mind was clear; and automatic beating of the forearm upon the bedcovers, convulsions with slight remissions, occurred in these cases. He believes to have aborted and removed this affection several times by means of applications of cold to the entire back of the patient. I myself have seen such a case in a boy aged four years. [I saw it in an infant of two years.—Ed.] The physician in attendance believed the affection to be a severe meningitis. The disease began with diarrhea and vomiting, without convulsions. At the examination, however, the contrast between the absence of stupor and the rigidity of the muscles of the neck was conspicuous to me. Also the inclination to take nourishment was astonishing. An examination of the lungs showed a pneumonia of the right apex. Upon the eleventh day crisis occurred, with complete recovery.

Conspicuous unrest, fear, shrieking at the approach of the physician or the nurse, I have seen in some few cases, not only, as Rilliet and Barthez have seen, in the pneumonia of children, but also in other affections occurring in childhood; for example, in enteric fever.

Steiner is probably correct in not allowing a single cause to give rise for the brain symptoms in children; he names: The prevalent disposition of the infantile brain in general to suffer in sympathy in inflammatory processes of other organs; the hyperemia of the brain from an increase in the temperature; the hyperemia of stasis of the meninges in the brain; the simultaneous occurrence of a purulent otitis; toxic action of the blood upon the central nervous system; results of reflex action; finally, a complicating meningitis.

Through the influence of rays of heat upon the head v. Jürgensen has seen severe brain symptoms which remind one very much of meningitis. In the case of two school-children a walk in the hot July sun was the cause of these phenomena. He ascribes these phenomena with great assurance to the influence of the sun's heat, as he had several times observed severe brain symptoms in the course of pneumonia of children whose heads were exposed to the rays of heat emanating from a very strongly heated oven. After this deleterious cause had been removed the cerebral symptoms disappeared.

In adults the functional implication of the nervous system shows itself by the occurrence of delirium. Most frequently they arise—also according to my experience—in apex pneumonias, and in such cases are especially long in duration. Huss believes that this symptom is not more frequent in pneumonia of the apex than when the disease is located at the base. These subjective expressions are, however, opposed to the results of the investigations of Heinze, which are based upon the reports of exact figures. Of 317 pneumonias, 98 showed severe symptoms on the part of the nervous system; and of these 98, in proportion to the seat of the pneumonia, 40.17% were at the apex and only 25.5% were situated at the bases. Why there should be such severe nervous symptoms in pneumonia of the apices Heinze also leaves undecided; only he declares it as certain—in contradiction to Liebermeister [156]—upon the basis of his material that the disturbances which arise in the function of the central nervous system in the course of pneumonia do not depend alone upon the rise in temperature; in fact, that in very many cases there is absolutely no connection between them.

[We have seen apex pneumonias admitted to insane hospitals for acute mania and acute mania believed to be apex pneumonia. One patient escaped from the nurse and ran in terror from delusions, a mile from his home.—Ed.]

Naunyn also is opposed to the opinion that the disturbances of the cerebral function which occur in so many febrile affections, especially those of the typhoid character, and which in these play such an important part, are due to an especial rise in temperature. The absence of these symptoms in relapsing fever proves the opposite of this view; also in the various febrile infectious diseases it is not unusual for the severest cerebral symptoms—for example, coma—to show themselves, whereas the temperature of the body may be only slightly raised, normal, or subnormal. Such conditions occur in scarlatina, variola, diphtheria, and pneumonia.

Further, it must be mentioned that chronic alcoholism is frequently the cause of **delirium**, which may occur and last during the entire duration of the affection. This delirium may be looked upon as a prodromal stage of delirium tremens, the condition not coming to the full point of development. Huss estimates the occurrence of this form of delirium in 10% to 12% of all cases. This estimate, however, cannot claim to be generally correct, as this symptom depends on the general spread and use of alcohol. I at least have not noted delirium with such frequency.

Formerly, under the treatment of pneumonia by venesection, delirium was noted much more frequently. One is therefore not surprised if in drunkards, after venesection, severe forms of delirium arise which may lead to conditions of maniacal delirium; as was actually the case in the observations of Lutz.

In a manner similar to the occurrence of delirium in the case of drunkards and after venesection, the form of delirium arising at the time of crisis or in connection with it must be referred to a condition of the nervous system which can be explained upon the basis of exhaustion. Rilliet and Barthez have occasionally noted very severe forms of delirium in children of from seven to ten years of age during the period of resolution; that is, between the seventh and tenth days. I myself have repeatedly noted, in adults whose nervous system during the attack did not show special implication, severe delirium after the crisis, and continuing for several days, with the entire absence of fever. The favorable action of the preparations of iron would substantiate my opinion that these forms of delirium were due to an exhaustion of the brain.

[Post-critical delirium or confusion, post-pyrexial delirium, has been frequently described: following the crisis, which may or may not have been associated with very marked temperature drop, in a varying time, sometimes at once, sometimes in say three to six days, a true delirium ferox may develop without any temperature increase; the delirium may last twelve to fourteen days and leave a mental unbalancing which may take some weeks to set right. A mild confusional delirium is at times seen, lasting but a few days and readily clearing up. In neither form is an antecedent alcoholic history to be expected. Whether due to excessive formation of "antitoxins" or the reaction, whatever it may be, which causes crisis, or to absorption of exudate, cannot be easily determined. (An article by Broadbent on delirium following a rapid absorption of exudate, thirty-six hours.) Similar insanities occur in other diseases (post-typhoidal). The majority of instances are in acute sthenic cases in which a decided crisis has occurred, and in which during the febrile period the mind has been decidedly clear.

Three cases have come under my observation recently:

1. Adult, aged thirty-five, non-alcoholic. Right apex. Crisis, seven to eight days. Tenth day wildly delirious, clearing up in four or five days. Afebrile. Leucocytes dropped with crisis. Perfectly clear in mind during febrile part of illness. Resolution proceeded normally. Pulse good; no collapse.
2. Adult, male, aged seventy-two. Twenty-four hours after crisis

violent delirium, with confusion and hallucinations. Continued one week. Sleepless. Hyoscin hydrobromate was of service.

3. Child, seven years. Right base. Crisis, six to seven days. Seventh, eighth, and ninth days slept continuously; quite irrational when roused; not violent, merely confused. Crisis, marked drop of 7 to 8 degrees in temperature; leucocytes dropped with temperature. Resolution normal and complete. Profuse perspiration and slight symptoms of collapse during crisis.—Ed.]

Hallucinations are more frequent than is generally believed. In an attack of mania with delusions of persecution combined with hallucinations, a man of thirty-eight who was suffering from pneumonia, and who up to that time had been quite rational, suddenly sprang out of bed, grabbed his night-table, and threw himself, thrusting the table before him, out of the second story of the ward through the window, which scarcely had a diameter larger than the table, smashing the entire window-frame. The nurse was scarcely ten feet distant, and almost before the nurse could move, the patient, in the sight of 20 other patients in the ward, had carried out the act. Incidentally it may be mentioned that the impression of this act was so terrible that the nurse declared himself incapable of attending to his duties any longer, and gave up his occupation.

To the province of symptomatic disturbances of the central nervous system, it appears to me there belong the **hemiplegias** which arise in the course of pneumonia. Stephan* has collected a list of his own cases as well as a list of those from other authors, and has endeavored to explain their causation. He first quotes the explanation of Lépine, which supposes that pneumonic hemiplegia occurs only in the aged, from which it follows that an atheromatous condition of the blood-vessels is the actual cause of the paralysis. Under normal conditions the circulation of the brain is sufficient for its nutrition, especially as there is often a compensatory hypertrophy of the left ventricle which markedly assists the circulation; as soon, however, as a disturbing element (a pneumonia) occurs, the force of the circulation is not sufficient to allow the brain to perform its normal functions.

According to this, the aged are predisposed to apoplexy; a trivial cause, a reflex irritation of the brain,—which may be produced experimentally in animals by irritation of the pulmonary substance,—is sufficient to favor the occurrence of hemiplegia; there is a paralysis, due to ischemia of the nervous tissue, which may be produced by atheroma of the cerebral vessels, and a reflex action having its origin from the diseased lung.

Stephan does not coincide with this explanation, because the origin of pneumonic hemiplegias in adults whose cerebral vessels are not atheromatous would not be explained. Such cases have been observed by him as well as by Macario. He assumes that, similarly to uremic hemiplegia, those cases of pneumonic paralysis arise in which the brain and the meninges show no anatomic changes, the pneumonia being produced by toxic substances perhaps belonging to the ptomains which are the cause of the paralysis, and that they, by means of reflex action, give rise to an ischemia of the opposite cortical centers. The atheroma, the fever, the deficient nutrition, a certain condition of cardiac weakness, may occur in the pneumonic infection, and must be looked upon as conditions favoring such paralysis.

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* The second case described by Stephan occurred in a child aged two, who succumbed to the affection. The frequent attacks of vomiting, the contractures which occurred in the further course of the malady, the ptosis and mydriasis of the right eye, the subsequent paralysis, the Cheyne-Stokes respiration, and the absence of a postmortem, make it impossible to decide whether or not a meningitis was present, and to say whether this case was identical with the one observed by me.

My own observations, occurring in two children aged one and three-quarters, and two and a half years respectively, allow me above everything to exclude a meningitis. The assumption of such a condition is impossible, as there was not a single characteristic symptom. Against this diagnosis was the absence of vomiting, of rigidity of the muscles of the neck, and especially the complete *restitutio ad integrum* which occurred, in the second case after a few hours, and in the first case after fourteen days. Therefore in these cases, as in cases occurring in adults in which there is no anatomic lesion of the brain or of its membranes, and in which none such can be assumed, another explanation must be sought for.

The occurrence of pneumonic hemiplegia in children, which I have observed, is against the possibility that in adults an atheroma of the vessel is of especial or principal importance. Only as an adjunct it should not be undervalued. The rapid recovery proves that a severe lesion, perhaps a hemorrhage or an encephalitic area, may have been the cause; it is then very obvious to assume, as Stephan does, that a disturbance in the circulation is at fault in this process.

But this explanation is not sufficient to make one understand the partial affection of one-half of the cerebrum. I tried to prove at the place referred to that in the same way as in uremia a partial edema of the cerebral substance in consequence of the changed constitution of the blood must be taken to be the cause of the hemiplegia. From this the fact explains itself as well as the favorable conditions for the *restitutio ad integrum*. In both cases rapid recovery took place.

If the occurrence of such hemiplegias in children is exceedingly rare, this may be explained by several circumstances. In the first place, one must assume a special disposition or feebleness on the part of the organ which suffers the complication, and this need not necessarily occur frequently. Furthermore, it seems to me that the seat of the pneumonia is not entirely without importance. In my cases the upper lobes were affected. If such pneumonias which are seated in the upper lobes are easily complicated with cerebral irritation in adults, this is the case in a much higher degree in children, but in these the irritations in the majority of cases occur in the motor areas. If we look into the causes which give rise to the frequent occurrence of cerebral irritations in upper lobe pneumonia, they need be explained in such a way that in these pneumonias the influence of the negative inspiratory pressure in the mediastinum is most reduced, therefore the reflux of the blood from the cerebrum to the heart is rendered more difficult. This difficulty, however, of the venous reflux from cerebrum to heart is found to favor extremely the secretion of serum from the arterial vessels or capillaries into the cerebral substance, principally in children. But it seems to me that the occurrence of apex pneumonias in children is comparatively more rare than that of the lower lobe, a fact which may confirm the less frequent occurrence of such hemiplegias in children. Finally, it is also possible that hemiplegias in children which occur in connection with apex pneumonia take so much predominance that pneumonias are overlooked in consequence, especially as the percutory and auscultatory signs in apex pneumonia are not absolutely characteristic, and pneumonic sputum is not produced—another reason for the rare observation of pneumonic hemiplegia in children.

In connection with the phenomena on the part of the nervous system **epistaxis** must be mentioned. It occurs frequently, is independent of the height of the fever, and of importance only when it occurs in the aged, in whom one must fear that the vessel-walls have become brittle, and the bleeding will be difficult to control. In such cases I have had to use a tampon in the nose.

Formerly great attention was paid, in both a diagnostic and a prognostic sense, to the condition of the **urine.** The importance of the changes in the urine, however, in the course of time has shown this to be unfounded. Only as regards the changes of metabolism of the body is the examination of the urine in pneumonia of value.

The *quantity of urine* during the fever is diminished, reaching or exceeding the normal after the crisis. The specific gravity is increased. The color of the urine is darker than normal; it is "high-colored." The reaction, as a rule, is markedly acid.

Exact investigations have been undertaken in regard to the behavior of *urea.* The results are unanimous as regards pneumonia in individual cases, that during the febrile period more urea is excreted than in the early period of convalescence, even after the organism has returned to its normal condition, by administering the ordinary foods and in the same quantities. Further, in many cases it has been shown that, with the onset of resolution of the pneumonia, the excretion of urea steadily increases compared with the previous days (Huppert, page 17).

Scheube gives several reasons for this epicritical increase of the excretion of urea: First, it is not unreasonable to believe that even after the defervescence the disintegration of albumin and, therefore, the excretion of urea for some time continue to increase. Secondly, the diuresis which sets in at the time of defervescence probably plays a part in the post-febrile increase in the urine, in that during the fever, as a result of the diminished secretion of urine, a part of the formed urea is retained in the body. According to Vogel's investigations, it is certain that the amount of urea increases and diminishes in direct proportion to the secretion of urine. Thirdly, one must assume that with the resorption of the exudate in the lungs a certain quantity of nitrogenous substance which has been kept out of the circulation is returned to the blood and now is subject to oxidation.

The latter condition is probably the most important circumstance in the increase of the epicritical excretion of nitrogen. If regard be paid to the previously mentioned (p. 387) mass of material contained in the alveoli, and the small amount of expectorated material as compared with it, one is forced to the conclusion that in a very short time an extraordinarily large amount of nitrogenous substance is returned to the circulation and undergoes oxidation.

The action of the *urates*, or, in other words, of *uric acid*, in pneumonia, has always been subject to special investigation; especially at the time of crisis the urine, after it has become cold, precipitates a copious sediment of urates, either spontaneously or after the addition of a few drops of acetic acid. This led to the error, which still exists largely among practitioners to-day, that the presence of a sediment of urates or crystals of uric acid in the urine, after it has cooled, is a sign of a plentiful amount of uric acid. But neither a sediment of urates in the urine nor the spontaneous separation of crystalline uric acid from clear urine proves a plentiful amount of these products (Bartels[15]), although Scheube thinks he has proved that in pneumonia, if the averages of several days, with the increase in the disintegration of albumin, are taken, the proportion between urea and uric acid decreases in favor of the latter. This author also emphasizes, in keeping with Bartels, that the separation of uric acid depends chiefly on the degree of acidity of the urine. According to Franz Hoffmann's method, he determined in the cases investigated by him that, for example, a urine 100 c.c. of which contained 113 mg. of uric acid and 182 mg. of acid presented the most favorable conditions for the production of sediment, whereas in another urine, with 122 mg. uric acid, and a total acidity of only 53 mg., no urate sediment was deposited. One could therefore artificially, through the introduction of phosphoric acid, produce urate sediments, whereas, on the other hand, through the taking of alkali the production of the same was decreased. If, however, the days upon which the defervescence occurs are especially characterized by a urine which deposits sediment, this finds its explanation in the fact that the disintegration of albumin, and, therefore, also the excretion of uric acid, increase with the onset of the fever. The conditions for the production of sediment, therefore, are more favorable upon the day of defervescence than upon the days preceding this event. Upon the day after the crisis the uric acid excretion is still more increased, but through the taking of foods and drinks of an alkaline reaction, the degree of acidity of the urine is lessened. After the defervescence, therefore, just as before, there is less frequently the production of sediments or urates, as upon the day of the crisis itself, at which time a high degree of albuminous change, without the use of food and alkaline salts, occurs.

The excretion of *sodium chlorid* in the urine in the course of pneumonia may fall to zero (Redtenbacher). This occurs here just as in all other acute febrile diseases. The cause of this very diminished excretion of chlorin is largely due to lack of appetite and to the nature of the diet of such patients, from which salt is practically eliminated. Also the decrease in the amount of urine on which this excretion is somewhat dependent decreases the amount of the chlorin combinations which are excreted (Neubauer and Vogel, p. 320).

Beale comes to the conclusion, upon the fact that pneumonia sputa contain a larger amount of fixed chlorids than the normal pulmonary mucus, that the decrease of chlorids in the urine during the stage of hepatization is due to the increase of these salts in the lung, whereas in the stage of resolution it is taken up and reappears in the urine. Traube declares this explanation is insufficient. Three facts are opposed to it. In the first place, such a decrease in chlorids also occurs in other febrile affections, which run their course without marked exudation. Further, the amount of chlorids in the urine in pneumonia bears no relation to the size of the exudation. In circumscribed hepatization one finds frequently larger amounts of chlorin than in large infiltrates; and, thirdly, one finds occasionally in the stage of convalescence of pneumonia, while the exudate is in the stage of resolution, that the amount of chlorin is just as small as it was at the acme of the affection. This phenomenon, therefore, cannot possibly have anything in common with the exudation. The more likely, therefore, is the fact that this change is connected with certain changes in the digestive apparatus, which may accompany pneumonia just as they do other febrile affections, and may produce, as a result, a diminished absorbing power of the gastro-intestinal mucous membrane. The condition of the convalescent patients referred to seems to substantiate this opinion. They took food containing chlorid of sodium in comparatively large amounts, but the heavy coating of the tongue and other gastric phenomena seemed to point to the fact that the digestive apparatus did not as yet functionate normally. The idea of making use of the amount of chlorin contained in the urine in a prognostic sense, that the prognosis was the more favorable, the more chlorin was contained in the urine, is valueless. It is said that in a case of a pneumonia just before death the urine contained an unusually large amount of chlorids.

Röhmann has investigated this question most thoroughly. Upon the basis of exact weighing of the food containing sodium chlorid in cases of pneumonia and the amount excreted in the feces and urine, he arrived at the result that the chlorids taken in as food were almost completely taken up by the bowel; that, according to this, a deficient absorption was not the cause of the disappearance of the chlorid of sodium in the urine; that, moreover, a retention of the same takes place in the body, which is the result of the general metabolic changes of fever, in pneumonia as well as in other febrile affections. Here a more or less large part of the tissue is disintegrated. Albumin of the tissues is taken up in the circulation. Of these, only a part is immediately transformed to its final products; the remainder, as the character of the excretion of nitrogen shows, during and after the defervescence, is retained in the body. In the plasma it combines with chlorid of sodium, prevents its excretion, and in this way produces the diminution of chlorids in the urine. Only if during and after the crisis the amounts of albumin retained are decomposed, and their nitrogen with the critical or epicritical excretion of urine leaves the body, then the chlorid of sodium becomes free and leaves the organism in the same amounts in which it was formerly retained. With this view as a basis, it is easy to explain why in some subacute febrile affections occasionally no retention occurs. Here in a long-continued fever a period must arrive in which the plasma has attained its maximum, in which it may take up albuminous substances, in which, therefore, no more chlorid of sodium can be used in combination; the retention disappears and just as much chlorid of sodium is excreted by the kidney as is taken up with the food.

[Hutchinson has studied chlorid metabolism in pneumonia. During acute lobar pneumonia the chlorids in the urine are greatly diminished, and may disappear altogether. Experiments in metabolism show that this diminution is due to retention, but the retention is not due to any lack of excreting power, since it does not occur in acute nephritis. The retention, the average amount being 29 gm., persists until a day or two after the crisis, when excessive secretion takes place. The degree of retention or diminution of chlorids in the urine bears no direct relation to the degree of pyrexia or the area of lung tissue involved, or to the presence or degree of albuminuria. Other solid constituents of the urine are not diminished in pneumonia.

The amount of chlorids excreted in the saliva in pneumonia is relatively large, but the daily amount is absolutely small.

Diminution of the chlorids in the urine occurs in other diseases as well as in pneumonia, and the sign is therefore not to be regarded as pathognomonic; but it does occur most constantly and to the greatest extent in pneumonia, and is therefore of distinct diagnostic value, especially in relation to other diseases of the lungs.

The quantity of chlorids contained in the exudate is not sufficient to account for more than one-third or one-half of the total amount retained during the course of a pneumonia.

The chlorids in the blood are diminished in pneumonia. They do not, however, accumulate in any particular organ, since all the tissues contain a higher percentage than under normal conditions. Hutchinson believes that this passage of chlorids from the blood into the fixed tissues is due to primary exudation of water.—Ed.]

The amount of *sulphuric acid* in the urine is decreased in inflammations of the lung. *Phosphoric acid*, however, at the acme of the disease is increased.

Peptonuria, which, according to the method of Hoffmeister, can be determined positively, has been demonstrated in the greatest proportion of the cases by Meixner, v. Jaksch, Pacanowski, and Müller. The appearance of peptone is connected with processes which have to do with the resorption of purulent exudates, or at least with exudates which are rich in cells; in pneumonia a sign that the pneumonic infiltrate is being absorbed (v. Jaksch, p. 431). These findings are not of especial clinical import, any more than the diazo reaction, which occurs in pneumonia, as well as in enteric fever, in phthisis, in measles, and in septic affections.

The **digestive organs** show their part in the affection, as in all febrile affections in which neither the stomach nor the bowels are the seat of the disease. *Vomiting*, which is especially frequent in children, occurring at the onset of the disease, is without doubt of cerebral origin, due to an irritation of the vagus. Rarely does vomiting in pneumonia continue beyond the first few days. Grisolle has seen this symptom last for four days.

Another cause of vomiting is due to severe paroxysms of coughing and the suffocative sensation combined with it. A previously present pharyngitis, especially in alcoholics, aggravates the condition. If this latter condition can be excluded, the occurrence of vomiting in the course of the disease points to the possibility of a severe complication.

The *tongue* is coated, and almost during the entire course of the affection *anorexia* is present. The taking of large amounts of liquid nourishment is nevertheless possible.

The act of *swallowing* frequently gives rise to tormenting paroxysms of cough, and sometimes even a disturbing thirst is not quieted because the patient fears cough. "It has occurred that it has been necessary to give a patient an injection of morphin in the region of the larynx (!) to quiet the irritation of coughing, and to force him to take a tablespoonful of an active and necessary medicine" (v. Jürgensen, p. 94).

The *bowels* are constipated in a majority of the cases, or else they are normal. Very rarely is diarrhea present, the latter perhaps as a result of the too great taking of fluids. [In some years *colitis* has prevailed to an unusual degree, at other times it occurs less frequently, but it is always a possibility and may become an alarming complication, giving rise to tympanites and causing exhaustion.

Abdominal pain may occur in pneumonia. The upper quadrants are usually the seat of pain if it occurs, although it may occur in the lower quadrants. The pain may be excruciating and simulate that of peritonitis, appendicitis, and gall-stones. I have seen cases about to be operated on for appendicitis turn out to be pneumonia. Especially is this liable in children, in whom vomiting also takes place frequently.—Ed.]

Liebermeister found the **spleen**, in contradiction to most authors, as a rule enlarged; occasionally even markedly enlarged. The **skin** shows few, but noteworthy abnormalities. With the onset of the affection *cyanosis* may occur, as has already been mentioned. It is, however, rarely marked; neither need it be looked upon as serious if there be no symptoms of edema of the lungs. In the further course of the affection a combination of both symptoms may occur, and influence the prognosis very unfavorably.

A frequent symptom is the occurrence of **herpes**. Among 421 cases of pneumonia, according to Geisler, 182 times herpes was noted; therefore in 43.2% of all these cases. In reference to the time of the appearance of this symptom, the same author shows that herpes occurs most frequently upon the third day of the disease, next upon the fourth, then follows the second, especially its second half, and then the fifth day of the disease. Within three days of the second day of the disease, the majority of cases of herpes therefore make their appearance. Occasionally resolution in pneumonia is ushered in by the appearance of herpes (Grisolle).

As a curiosity I must mention an observation I made on my colleague S., whom I met upon the street, and in whom I noticed the appearance of herpes upon his upper lip, and asked where was the pneumonia that belonged to this condition. Two days later he was affected by a very severe pneumonia, from which he recovered.

The locality of herpes is usually in the neighborhood of the mouth and the nose; usually at the point at which the mucous membrane joins the skin. It may, however, occur in other places. Thomas saw an eruption of herpes 3 cm. from the anus; 50 to 60 vesicles covered a round space about the size of a dollar. [Herpes of the nose, lips, chin, and ears occurred in one, and of the nose, lips, chin, and legs in another, of 26 cases in which I saw it.—Ed.]

Perspiration may occur during the entire course of pneumonia (Grisolle, Traube). In children, whose skin, as a rule, is hot and dry, occasionally at the height of the fever a profuse perspiration is noted. One may note that if the children fear the examination of the physician and become excited, especially if they markedly resist, perspiration easily occurs (Baginsky). With the defervescence and the occurrence of the crisis, however, perspiration is especially frequent —most frequent in youthful individuals and in an uncomplicated case. The amount of secretion of the sweat-glands may occasionally be so great that body and bed-linen are completely wet through.

As a result of such severe sweating marked sudamina, in the form of miliaria rubra and alba, may occur.

Profuse perspiration may also occur before or at the beginning of the agonal period. The skin, however, is then cool, cyanotic, the temperature nearly always very high, the pulse small and frequent, and the brain clouded.

Wunderlich has made some observations in reference to **attitude** in pneumonia patients. They usually lie quietly upon their backs.

The position upon the side is uncomfortable; upon the sound side it is more uncomfortable than upon the diseased side. In the case of severe pain they never attempt to lie upon the diseased side. The erect posture is very uncomfortable. This is at least true of pneumonia of the bases, in which the diseased part always takes up the posterior space of the thorax, and therefore the position upon the back is the least detrimental to the rest of the lung, the diaphragm, or the heart. In pneumonia of the apices and in disseminated lobular pneumonias, on the other hand, the side position and the erect sitting position are used to relieve breathing (Wunderlich [258], vol. III, p. 476).

THE COMPLICATIONS OF PNEUMONIA.

Beginning with the lung, I shall retain the order employed in reviewing the symptomatic implication of the other organs, and shall not describe the complications of pneumonia in the order of their frequency.

[The complications of pneumonia are really expressions of the pneumococcus infection in other organs or tissues. Hence, in addition to pleurisy, pericarditis, endocarditis, meningitis, and nephritis, mentioned by the author, we also see arthritis, phlebitis, colitis, peritonitis, otitis, parotitis, conjunctivitis, and general mucous membrane infections, as in the cases of Cary and Lyons.—Ed.]

To the rarer events belong **abscesses of the lung.** Laennec, at the time of the first publication of his book upon auscultation and percussion, among several hundred autopsies of pneumonia noted only "five or six cases," and only once an abscess of considerable size (vol. I, p. 406). Into this scarcely the tips of three fingers could be introduced. Andral found abscess but once, in a patient who died on the nineteenth day of a pulmonary inflammation. The description which Andral gives of this abscess is characteristic of the great majority of such cases; it deserves, therefore, to be quoted verbatim. The condition in question related to gray hepatization of the middle and lower pulmonary lobes. The center of the latter contained a dirty gray mass in the middle of which actual pus with no especial odor was found. Surrounding this the pulmonary tissue was very soft, in a condition of detritus, and gradually took on a firmer consistency. During life there were no signs of the presence of an abscess.

I myself have found among 253 fatal cases of pneumonia (202 men, 51 women), which were nearly all examined postmortem, three abscesses of the lung. Once the abscess was combined with an empyema; the anatomic condition was just as Andral described it. [It occurred in two of my 195 cases.—Ed.]

If Lebert, in the four cases of pulmonary abscess following pneumonia which he has observed, found the cavities small, of a caliber varying between the size of a hazelnut and that of a walnut, and clothed with an organized pseudomembrane ([143], p. 647); it must have occurred in a very late stage of the process.

Grisolle has collected 29 cases from different authors, and finds that the abscesses are usually near the surface, that they measure from 3 to 15 cm., have an irregular sinuous outline, and that their walls are bounded by shreds of tissue. In rare cases an abscess membrane is present. Once the abscess had broken into the pericardium. Of the 27 abscesses, 11 were found in the right, and 16 in the left lung.

In 25 cases the age was given. According to the ages there were:

	at	4	years	1	abscess.
between	16 and	26	"	4	abscesses.
"	31 "	36	"	5	"
"	45 "	49	"	3	"
"	50 "	58	"	4	"
	at	70	"	8	"

As Skoda has quite properly remarked, the diagnosis of abscess of the lung cannot be made by auscultation and percussion alone. "A probable diagnosis might be made if with the rapid increase of the pneumonic symptoms a large quantity of pus, colored red by blood, should be brought up at once, and should be followed by improvement in the pneumonic phenomena; certainty can only be given after the demonstration of elastic elements of the parenchyma of the lung in the sputum." Better points for the diagnosis of pulmonary abscess are given by Traube ([238], vol. I, p. 483). In acute pneumonia suspicion of an abscess is raised if resolution of the inflammatory exudate is slow, and the fever, which occasionally is only slight, lasts longer than usual. Of especial diagnostic value, however, is the sputum. This is usually very copious, forming a mass only slightly different from the usual connective-tissue pus, in which but little mucus is contained, and which, as a rule, has a musty purulent odor—it may, however, through longer stagnation of the pus, become fetid—and macroscopically contains particles of parenchyma of the lung, of irregular villous appearance, of black-spotted, rarely yellow, color, whose length varies from that of a millet-seed to 6 cm. (1.20 inch). The microscopic examination of the particles shows an abundance of elastic tissue with free black and yellow pigment, which latter is partly amorphous and partly crystalline. Only particles of parenchyma of the lung of this nature are characteristic of abscess, for Traube has seen a case of abscess in which the sputum, except for these shreds of lung tissue, presented nothing unusual either in appearance or in quantity.

Pulmonary abscesses may heal completely. Two such cases are explicitly described by Traube. The finding of many elastic fibers in the sputum made the diagnosis positive.

Pure necrosis of the pulmonary tissue I once saw. It occurred in a case of metapneumonic empyema. The pus was emptied from the pleural cavity by resection of the rib. Some days after this had occurred, in washing out the cavity, two particles of tissue the size of a plum crowded themselves into the opening at which the resection had been performed and could be readily taken out. The microscopic examination confirmed the macroscopic appearance, that the particles consisted entirely of pulmonary tissue. The patient was discharged cured.

Somewhat more frequently pneumonia terminates in **pulmonary gangrene**; but the condition must be looked upon as rare in comparison with primary gangrenous inflammation of the pulmonary tissue. Grisolle, in the 305 cases of pneumonia upon which he bases his report, did not note one in which gangrene occurred, and in an examination of 70 cases of pulmonary gangrene which were described by various authors he scarcely found five which, speaking strictly, could be taken as examples of pneumonia terminating in gangrene of the lungs. Not much greater is the number observed by Hensel in the Breslau Pathological Institute. In his Table III, only 5 among 73 cases of gangrene can be referred to croupous pnumonia.

The diagnosis "croupous pneumonia terminating in gangrene" may be made only when, after all the characteristic symptoms of pneumonia have been present for several days, signs of gangrene are added. Convincing cases of this kind have been reported by Kannenberg, Leyden[154], and others. In cases in which the result is favorable the cure of gangrene of the lung can only be accepted with certainty if elastic fibers have been seen in the sputum. Without these, putrid bronchitis might easily be mistaken for this condition. Such putrid bronchitis, however, I have repeatedly seen in pneumonia, especially if simultaneously an empyema existed. "The sputum in putrid bronchitis has the same repulsive smell as in gangrene of the lungs," says Hoffmann ([103], p. 146). In my experience, this may at least occur a few times. Mostly, however, putrid bronchitis may be easily differentiated from gangrene by the difference in smell.

The most frequent complication of pneumonia is **pleurisy**. [In 55 of 195 of our cases.—ED.] As has previously been emphasized in the description of the pathologic changes, there exists in every pneumonia an inflammatory change in the contiguous pleura. As this complication, however, in a majority of the cases is not an independent affection, and, moreover, disappears with resolution of the exudate, it might be well to speak of a complicating pleurisy only if the condition lasts longer than the pneumonia and requires especial treatment. Very likely, upon the different definitions of this condition the unequal size of the different reported figures depend. According to the statement of v. Jürgensen, the number of pleurisies complicating pneumonia in Vienna were 5.2%; in Stockholm, 4%; in Basel, 15%. According to my observation, in 1501 cases of pneumonia 59 serous pleurisies occurred, 24 empyemas, together with 83 affections of the pleura; therefore these occurred in 5.5% of all cases.

As a rule, the pleurisy occurs at the end of the pneumonia. The pneumonia may end by crisis, and soon thereafter fever may occur which is dependent upon disease of the pleura. There is scarcely, however, another affection which shows such extraordinary variations in regard to fever. I have observed high remittent fever with serous effusions; and I have known entire absence of all rise in temperature for ten days, in cases of very large empyemas—that is to say, absent up to the time of operation and afterward..

Regarding the diagnosis, an exact description will be entered into in the section especially devoted to pleurisy; here there need only be mentioned in regard to prognosis that, according to my experience, the complication of a serous pleurisy or an empyema only increases the duration of the attack. The prognosis in serous effusion is not made markedly worse, whereas in the case of a purulent effusion, through the possibility of several conditions at and after the operation which cannot be determined beforehand with certainty, a favorable result cannot in the same positive manner be predicted.

Among complications occurring in other organs, as regards danger, but not as regards frequency, those relating to the pericardium and heart take the first place.

Inflammation of the **pericardium** is similar to that of the pleura, and may be either of a serous or of a purulent nature. In the latter case a simultaneous purulent inflammation of the pleura, with a more or less abundant fluid exudate, may be present. A rare case of this kind, occurring after the subsidence of a pneumonia, was admitted to this hospital (Breslau). A right-sided empyema in a man aged thirty-four was emptied by means of resection of the ribs, but three days later death occurred. At the autopsy another completely encapsulated empyema of the mediastinum was found between the inner surface of the right lung and the pericardium, as well as a purulent pericarditis, with slight accumulation of free pus in the pericardium. Another noteworthy case of purulent pericarditis in connection with pneumonia occurred in a child eight months old. This case also terminated fatally.

The method of treatment in pneumonia seems to be not without influence upon the occurrence of a pericarditis. According to the reports of Dietl, it must be assumed that venesection in a high degree favors the occurrence of pericarditis. This deserves to be especially emphasized, as at this time there appears to be a scientific revolution which is endeavoring to gain new friends for the therapeutic application of venesection, which has nearly entirely been done away with. Pneumonia must almost exclusively remain a *noli me tangere* for venesection. It would be useless to demonstrate all the unfavorable experiences anew. If there has been any point in the question of venesection which has been definitely solved by Dietl, it is that which clearly defines the influence of venesection in reference to pericarditis. His own words are: "From a general review of the various complications of pneumonia dependent on different methods of treatment it should not be difficult to judge of the influence of venesection upon the occurrence of complications in pneumonia. It is conspicuous that in 17 cases, after treatment with venesection, in patients who had died, pneumonia was complicated three times by meningitis and five times by pericarditis. On the other hand, in 22, after treatment with tartar emetic, in patients that had died, only one; and in 14 after treatment with dietetic measures in persons dying, not one was noted. It is not easy to assume that this frequent complication of pneumonia treated with venesection is accidental, as it occurred only in treatment by venesection, and in no other method of treatment. We believe, therefore, that we may allow ourselves the conclusion that the frequent complications of meningitis and pericarditis are caused by venesection, as we have proved that the coagulation of the blood favors the acute exudative processes and the extension of a hepatization from one lobe or part of the lung to another. This comparative postmortem finding seems to us to be one of the most important and memorable ones, and to substantiate our former statement that pneumonia grows and flourishes under the lancet. The more we practise venesection, the more atonic the capillaries of the lung become. The more coagulable the blood, the more fluid its fibrin becomes, and the more inevitable are greatly distributed hepatization and also infiltration of other tissues, especially the membranes of the brain and the coverings of the heart, until finally death occurs through enfeeblement of the blood and paralysis of the nervous centers. If, however, venesection does not cause this in all cases, it only proves that there are conditions of the blood that are capable of withstanding any influence up to a certain degree."

Endocarditis arising in the course of a pneumonia appears to vary as to time and location. I myself have seen but a single case of acute endocarditis affecting the aortic valves in the course of pneumonia. Fismer has in only two of his cases, therefore in 0.9%, seen acute changes at the mitral valve. Weichselbaum must have had occasion to examine such cases more frequently. He was in the position, by means of using pure culture and animal experiments, to demonstrate Diplococcus pneumoniæ upon the growths on the valves, and to substantiate the investigations of Netter, who, in nine cases of "*endocardite pneumonique,*" found seven times the same bacteria in the vegetations upon the valves as in the pneumonic lung,—namely, long diplococci with well-stained or unstained capsules,—but who was not in the position to grow cultures, or, through experiments upon animals, to establish the identity of the bacteria which he microscopically proved to be present in endocarditis with pneumococci. The latter Weichselbaum was able to do six times in 39 cases of endocarditis, in all of which he made careful bacteriologic investigation. In four of these cases there was pneumonia in addition to endocarditis. Here the question may arise how this variety of bacteria found its way into the blood and so reached the valves of the heart. It may be easily answered that the diplococcus of pneumonia, from the inflamed lung in which it was already present, found its way there. In one of the remaining two cases a purulent cerebrospinal meningitis was present. In the others, however, there was no process to which Diplococcus pneumoniæ might have given origin. But in this case also the possibility that the cause of the endocarditis found its way from the respiratory tract into the organism cannot so easily be put aside.

As regards the variations which the *endocarditis pneumonica* shows from the other varieties of acute endocarditis, Weichselbaum remarks that these may arise in the ulcerative as well as in the verrucose form, although, as it seems, more frequently in the first form. At least, in his cases the ulcerative variety was noted six times and only once did the verrucose form occur (here a case that was not counted above has been reckoned in).

The affection appears to show a special preference for the aortic valves—a statement already made by Heschl and Netter. In Weichselbaum's cases the aortic valves were affected five times and only twice was the mitral valve the seat of the affection. Moreover, this form is distinguished very frequently by the fact that very large and even occasionally polypoid vegetations form upon the valves; infarcts are not so rare as Netter supposes. Among the seven cases of Weichselbaum this occurred five times.

[Da Costa* reported three cases of phlegmasia alba dolens in the course of pneumonia. In two cases the veins of both legs were affected. This complication is rare, as but nine references in the literature of the subject could be found.—Ed.]

Next to the affection of the pleura, the most important complications of pneumonia in a numeric as well as in a prognostic sense are the complications on the part of the nervous system.

The frequent occurrence of **meningitis** in the course of pneumonia has been emphasized by Immermann and Heller on the basis of the earlier reports in literature; these authors also insist that clinically the symptom-complex is occasionally not at all characteristic.

Nauwerck has analyzed 29 cases of meningitis occurring in the course of pneumonia (23 men and 6 women). The complication occurred 7 times in the stage of red hepatization, 19 times in the stage of gray and yellow hepatization. Among the last 19 cases the pneumonia was noted 14 times to be in the stage of diffuse, well-developed pus formation. Once an abscess of the size of a pigeon's egg occurred, and twice there was beginning abscess formation. In three cases exact reports were missing.

As regards position, he determined that the convexity of the brain alone was affected 4 times; convexity and base, 16 times; convexity, base, and spinal cord, 7 times.

The meningitis occurred upon the:

3d day	once.	9th day	three times.
4th "	once.	10th "	six times.
6th "	four times.	12th "	twice.
7th "	once.	17th "	twice.
8th "	twice.		

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* *Phila. Med. Jour.*, Sept. 18, 1898.

In the remaining clinical histories exact reports are wanting.

Huguenin supposed that purulent meningitis in pneumonia in all cases was due to a purulent resolution of the pneumonic exudate. Nauwerck says that experience contradicts this. It must be admitted that in two of the cases in which the pneumonia was found in the stage of pus formation, decaying thrombi were found in the pulmonary veins. In one of these they were even puriform. The assumption is easy to believe that a mixture of a puriform degenerative material gaining access to the arterial blood stream, and thence being carried to the pia, will result in a consequent purulent inflammation.

In the main, according to Nauwerck, we have to do with an embolic infection with consequent purulent inflammation, and this infection arises from material which is derived from the diseased pulmonary parts and gains access to the arterial blood stream.

The opinion of Weichselbaum that the pneumococci reach the meninges by means of the cellular tissue of the neck, and there become the cause of the inflammation, has been mentioned previously and discussed more thoroughly. Whether this opinion, though, will hold for all cases remains questionable. In considering the facts of the existence of the diplococcus in the blood of pneumonia patients, proved by a great number of thoroughly reliable investigators, the fact that the occurrence of meningitis is limited to a variable but comparatively small percentage of all pneumonias must be ascribed to local conditions, which permit the localization and increase of the pneumococcus in the meninges. Here endemic circumstances may play a great rôle. If Meyer reports from the Pathological Institute of Dorpat that in 11 cases of typical croupous pneumonia, in which autopsy was performed, purulent meningitis was found as a complication five times, this would be an extraordinarily high percentage. I myself in the 253 cases, in almost all of which an autopsy was performed, found meningitis but 7 times; besides this, I have noted meningitis 3 times as a complication of pneumonia in patients who recovered. According to this, among my 1501 cases of pneumonia there were but 10 cases of meningitis.

Cerebral complications of the pneumonic process occur most frequently in alcoholics and give rise to the symptoms of the condition known as **delirium tremens**. By means of the primary affection —that is to say, by the blood taking up deleterious substances and conveying them to the brain—an irritation is produced which adds an additional irritation to that already present produced by the use of alcohol. In reference to the pathogenesis of this condition more accurate statements cannot be made at this period.

The frequency with which delirium tremens occurs in the course of pneumonia depends largely upon the distribution of the alcoholic habit in the community in general. Among Huss's patients no less than 180—that is, 6.91%—suffered from delirium tremens. Of the total number, 174 occurred in men and 6 in women. Among my

1501 cases, 80 were attacked by delirium tremens—that is, 5.3%; among these there were 76 men and 4 women.

As a rule, this complication is well developed at the acme of the pneumonia, somewhere about the third day of the affection. Great unrest and insomnia, which are not dependent upon the usual causes, —for example, cough or pains in the side,—and which, taken in connection with the previous history of the patient, make the diagnosis easy, usually precede the condition. Taking these facts into consideration, however, allows us to take the proper precautions and apply the proper remedies so as to lessen the dangers which this complication is liable to bring forth. This danger is grave, according to our observations made up to the present time. Huss lost among his patients, of those that had delirium tremens, 20%; Fismer, who reports only 12 cases, lost 50%; I lost in my 80 cases, 22—that is, 27.5%. That therapeutic precautions, however, are not without marked influence upon the recovery of such cases I can emphasize by the fact that, since the year 1891, of those cases that were treated in the manner to be described later, among 13 cases only 3 terminated fatally. One of these fatal cases was complicated by an acute nephritis.

Occasionally the entire brain is affected, without there being any predisposing cause, such as alcohol in delirium tremens. This previously undescribed condition of the brain I should like to designate as an *acute diffuse encephalitis*. It is true my observations are limited to but 4 cases, of which only 2 were observed in the hospital. The severity of the affection, the gravity of the danger, the possibility of obtaining therapeutic results, lead me nevertheless to go somewhat into details.

In these cases, besides the characteristic symptoms of pneumonia, at the height of the affection there was added the most marked degree of unrest. There was also absolute insomnia. The patients talked almost continuously, without necessarily being delirious; were restless, throwing themselves from side to side as if they could not obtain rest in any position. In one of these patients the unrest increased to such a marked extent that he broke out into almost continuous screams; with this he beat his chest with his fist, using all his force. It was almost impossible to prevent this. With all this the patients were not aggressive; they remained in bed. Two of these four patients died of exhaustion, it being impossible to quiet them. Only in one of these patients, the one who showed the marked unrest, was it possible to procure an autopsy. The result of the postmortem showed, besides a pneumonia involving the entire right lung, a wet brain and a high degree of hyperemia of the brain, with a conspicuously pink appearance of the cortical substance.

In regard to etiology, it appears to me to be worth mentioning that in both the cases which I had an opportunity to observe in private practice very marked and continuous anger preceded these phenomena —in one case combined with the loss of fortune.

Whether *focal affections of the brain* occur in connection with or in the course of a pure pneumonia appears to me to be questionable. While Isager regards as the result of a focal affection of the brain an aphasia which occurred suddenly with the crisis in a boy aged nine, and disappeared after lasting fourteen days, I should rather class this with the cases of paralysis in children previously described (page 477), and attribute the origin to the same causes.

Affections of the **spinal cord** are very rare in connection with pneumonia. Occasionally infantile paralysis has been seen as a result of pneumonia, and so described (compare Leyden[151]). Furthermore, Babés and Vellan have seen develop in a case of right-sided pleurisy and in a case of right-sided pleuropneumonia an atrophy of the muscles of the right half of the thorax, of the pectoralis major, the deltoid, the supra- and infraspinatus, the serratus anticus major, and all of the intercostal muscles of the right side. In the excised parts of the muscles disease of the finer ramifications of the nerves was demonstrated.

Up to the present time it was believed that **nephritis** as a complication of pneumonia was very infrequent. The finding of albumin in the urine was looked upon as febrile. Leyden says: "As frequently as febrile albuminuria is encountered in pneumonia, so rarely is a true nephritis encountered. When this, however, does occur, it develops as a rule from the febrile albuminuria and shows itself in the form of an exquisite hemorrhagic desquamative nephritis." The majority of such cases, he noted, terminated in recovery, although they lasted several days or weeks longer than the pneumonia.

Lecorché and Talamon noted the development of a hemorrhagic nephritis after fifteen hours following the initial chill in a case of pneumonia.

A very careful and exhaustive examination in reference to the connection between nephritis and pneumonia has been conducted by v. Kahlden, who carefully consulted the literature of the subject. In opposition to the opinion that an inflammation of the glomeruli with degenerative processes of the glomerular epithelium takes place, he found that in a certain number of the cases the epithelium of the convoluted tubules and of Henle's loops was implicated almost exclusively, and in those cases in which, in fact, the epithelium of the glomeruli was affected, the changes as compared with those in the convoluted urinary tubules were always of lesser significance. If in many of these descriptions the hemorrhagic character of this variety of nephritis is so markedly emphasized that it appears to be almost specific for pneumonia, or even pathognomonic, these reports are based upon the examination of the urinary sediment. In those cases in which histologic examinations of the renal tissue have been reported, the blood has been rarely found in the interstitial tissue, but mostly in the glomerular capsule and in the urinary tubules. Von Kahlden himself missed these hemorrhages in many cases, and in other cases they were never so numerous or so marked as had been reported by the majority of the authors.

In a pathogenetic relation he quite properly expresses the opinion that in the case of febrile albuminuria there was a change in the kidney structure which is actually the beginning of an acute nephritis, with which, from the same etiology, it is combined. This etiology depends upon toxic substances circulating in the blood.

Nevertheless it will remain justifiable to differentiate between the onset, therefore between a beginning nephritis and a developed nephritis, and to draw conclusions from a diagnostic and prognostic standpoint, and to give to the former as a rapid and transitory process the designation "albuminuria," but to take from it the idea that it is due to a febrile process. It is not difficult to determine the limits. If at the time of crisis the albuminuria desists, a nephritis has not been developed. Then, also, the quantity of albumin has been small. If we endeavor to reason in this manner, then the highest percentages which have been given of well-developed nephritis are not too low, as v. Kahlden supposes. My own observations do not even give so high a percentage as those of Wagner and Rosenstein. The former saw, among 150 cases, nephritis 4 times—therefore 2⅓%; the latter, in 130 cases, twice nephritis—therefore 1.7%. I noted, in 1501 cases, nephritis 16 times—therefore 1%.

Lately Fraenkel and Reiche have carefully investigated the condition of the kidney in pneumonia. In the kidney of pneumonia patients in adults there were: (1) The changes were almost exclusively limited to the cortical layer. Only comparatively rarely were casts in varying numbers found in the collecting tubules and in the terminal tubules; once these were partly occluded by granular material; otherwise, however, the epithelium, except for occasional deposits of pigment, was not degenerated. The connective tissue of the medullary substance never presented recent alterations. (2) In the cortex, with but few exceptions the secreting parenchyma, the loop of Henle, and the straight canals of the medullary rays were alone affected. (3) In every case there were found, although in variable amount and extent, exudates in the glomerular capsules.

The combined occurrence of **jaundice** and pneumonia has led to many varied controversies. Chomel fully described the differences existing in various combinations of pneumonia and jaundice, and pointed out that true icteroid pneumonia must not be confounded with that form of pneumonia in the course of which jaundice may arise, which may be due to a mechanical obstruction of the flow of the bile in the ductus hepaticus or choledochus or that may arise from a severe psychic disturbance—fright or anger, etc. In such an instance the icterus is accidental and is in no direct connection with the pneumonia. In the case of icteroid (bilious) pneumonia, on the other hand, the inflammation of the lung and the condition of the bile form an entity.

Mosler, on the other hand, believes that all combinations of jaundice with pneumonia are identical. He calls attention, in the first place, to the communications of Traube in reference to the frequent occurrence of the so-called bilious pneumonia, which distinguishes itself especially by severe symptoms on the part of the nervous system, even diminution of sensibility, occasionally tendency to diarrhea; he then adds a complete report of the earlier observations of Hauff, Potter, and Drake, and gives an exact description of four cases of bilious pneumonia terminating fatally. He attributes special value to the postmortem findings on account of the simultaneous occurrence of a gastro-intestinal catarrh and croupous pneumonia, which are important for his deductions. From this the conclusion may be drawn that the variations which bilious pneumonia show from the ordinary croupous pneumonia are due to the presence of a resorption icterus, and cause a mixture of the blood with bile acids. The degree of variation and the difference of the course depend upon the amount of the bile acids absorbed, the greatness of the obstacle in the biliary ducts, and the duration and intensity of the jaundice.

The necessity of separating the different varieties—that is, the older opinion of Chomel—has lately been emphasized by Bettelheim. He proposes for the one species of pneumonia which Traube, Mosler, and their predecessors had described, and which, according to their reports, runs a severe course and frequently terminates fatally, since the cases are complicated by jaundice, the name *pneumonia biliosa;* on the other hand, for all cases of pneumonia running their course with jaundice, he proposes the term *pneumonia cum ictero.*

[The consensus of opinion seems to be that there is no direct relation between pneumonia and the occurrence of jaundice, which is simply an accidental complication, more likely to occur in alcoholics and in those afflicted with some hepatic disease.

Petrov* says that the absence of jaundice during the period of resolution has convinced him that the condition bears no relation to the absorption of the exudate, and that compression of the bile-ducts has nothing to do with its production. Certain animal experiments which he performed lead him to believe that the jaundice is due to disease of the biliary passages and is not due to hemolysis.

Gilbert and Grenet † accuse Bacterium coli commune of producing an angiocholitis.—Ed.]

According to my opinion, I must entirely coincide, basing this upon my experiences, but must add that to genuine croupous pneumonia, whose cause is Diplococcus pneumoniæ, an icterus is added only when a mechanical occlusion of the biliary passages complicates the disease. We have to do in such cases always with a *pneumonia cum ictero.* In accordance with this is the fact that in a case of genuine croupous pneumonia a jaundice that appears does not necessarily influence the prognosis unfavorably. Of my 1501 cases, 15 were complicated by icterus. [It occurred in five or 2.6% of my cases.—Ed.]

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* *Gazette hebdomadaire de médecine et de chirurgie,* Sept. 9, 1897.

† *Archives générales de médecine,* February, 1899.

Of these, 2 died; and one of them had, besides, a chronic nephritis. Where, however, a bilious pneumonia arises, not Diplococcus pneumoniæ, but other bacteria are the cause; the bacteria are able at the same time to produce a pneumonia and a degeneration of the parenchyma of the liver, and to the latter cause the production of jaundice is due. [In Norris' series of 500 cases, jaundice occurred 18 times with a mortality of 61%.—Ed.] Such pneumonias are very much more dangerous to life. Their description does not belong to genuine croupous pneumonia, but to atypical pneumonia, which will be described further on.

[Arthritis is a complication or incident in the pneumococcus infection which may precede the lung infection or follow after the latter has subsided. Cave * has studied the reported cases of *pneumococcus arthritis.* Thirty cases were reported; 23 of the 30 died. Two occurred in children, the others in middle or advanced life. Suppuration of the joints took place in all but three of the cases. In many, pericarditis and endocarditis or a pleuritis accompanied the process. The joint complication began a few days to two weeks after the initial chill when it accompanied pneumonia. In some rare instances it preceded the pneumonia, and in a few cases pneumo-arthritis occurred without pulmonary infection.

The onset in the course of pneumonia may be indicated by an increased rise in temperature or the occurrence of a chill. If it follows the crisis, a chill and fever mark an invasion.

Miller reported to the College of Physicians of Philadelphia a well-marked case. Allen and Lull † reported a case of primary pneumococcus arthritis of the knee-joint, the fourth on record.

Concerning the *mucous membranes,* Carey and Lyon report a case in which during the course of a lobar pneumonia a croupous inflammation of all the mucous surfaces of the body took place, including the mouth, pharynx, and colon.

Labby and others have reported cases of parotitis, the complication indicating a grave infection.

Symmetric gangrene following pneumonia has been observed by Dufour.‡ The case was one of double pneumonia in a woman fifty-eight years old. Some days after recovery, generalized polymorphous erythema; then gangrenous patches on fingers, toes, nose (tip), lobules of ears. No preceding local asphyxia. From the first, vesicles and purulent fluid; albuminuria in excess; death from infection. Never any cessation of peripheral arterial obstruction. Blood (bacteriologic examination) negative; pus in left knee; peripheral nerves normal; tissue showed leucocytic infiltration; arteries not obliterated; veins swollen and inflamed. Dufour considers the affection to have been toxic, favored by less resistant powers of periphery and impairment of circulation. Peripheral symmetric gangrene has been produced in animals during experiments in which collodion capsules filled with cultures of micro-organisms have been introduced into the peritoneal cavity.

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* *London Lancet,* Jan. 11, 1901.

† *Annals Surg.,* Oct., 1901.

‡ "Soc. Med. des Hôp. de Par.," Oct. 18, 1901.

We may also have spontaneous gangrene consecutive to pneumonia. Tuppinger* reports a case in a child. On day after crisis pain in right great toe; two days later whole foot livid, and similar areas on left foot, right knee, and elbow. Two months later following lesions present: Ulcer on back, 10 × 9 cm.; anterior part of left foot completely disorganized; right great toe gangrenous; second and third toes also affected. Right popliteal pulsation absent. Left foot amputated. Good recovery. This is the first recorded case of post-pneumonic gangrene in a child.—Ed.]

Of other complications there must still be mentioned the appearance of **erysipelas** in the course of pneumonia. I can only look upon this as accidental. In my 1501 cases it occurred but five times, once with a fatal issue. In the latter case there was at the same time cirrhosis of the liver. Among the four that were cured, two had, besides, delirium tremens.

The presence of **angina** I have noted six times at the onset of **pneumonia.**

PNEUMONIA AS A COMPLICATION OF OTHER DISEASES.

The lungs in which pneumonia arises may have formerly been the seat of a chronic bronchitis, of emphysema, of a tuberculosis of the apices. **Emphysema** increases the danger to a marked degree. In tuberculosis pneumonia does not often arise. If I look back at the positive cases determined by autopsy, the number amounts to seven. In the presence of old **valvular lesions** I have, remarkable to relate, not had so high a mortality as is usually supposed. Of all the 13 cases, 3 died. Clinically these cases were marked by more or less well-developed cyanosis.

On the other hand, the presence of **chronic nephritis** brings great danger in regard to the prognosis of pneumonia; yes, it makes the impression upon me as if chronic nephritis even favored the production of pneumonia. Among my 1501 pneumonias, 22 cases of nephritis occurred; of these, 17 died.

Pneumonia occurred four times in patients suffering from **cirrhosis of the liver.** In all four cases the *exitus letalis* occurred.

The occurrence of pneumonia in **enteric fever** has given rise to much discussion. Hoffmann in particular made a number of thorough investigations. He determined that in enteric fever lobar pneumonia most frequently occurs at the acme of the typhoid process, or at the beginning of convalescence. It usually complicates a well-developed or severe enteric affection, with extensive alterations in the lymphatic apparatus of the intestine. In such cases the pulmonary affection is a very unfavorable complication, as it usually causes a decided rise of temperature and the result is more apt to be unfavorable. Nevertheless, even such cases frequently recover.

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* *Wien. klin. Woch.*, 1899, No. 13.

Besides such cases, others are also noted in which the pneumonic affection develops early. In such cases the bowel affection is frequently not very well developed. Whether such cases, which have been designated *pneumotyphus*, are to be included in the term enteric fever, must be decided by the behavior of the intestinal canal. If there are found in the intestinal canal at any place the characteristic typhoid changes, even if only very slightly developed, the case in question must undoubtedly be counted as enteric fever; should these changes, however, be absent, and the course of the affection resemble enteric fever as much as possible, we have to do only with a pneumonia running a typhoid course, not with a case of pneumotyphus that is to be regarded as belonging to enteric fever.

According to our present knowledge, the demonstration of typhoid bacilli might be utilized in the differentiation of these conditions, which is perfectly justifiable. In the last-mentioned group of cases, particularly, the presence of typhoid bacilli would have to be substantiated to prove their connection with enteric fever. More easy would the proof be if the diagnosis of enteric fever by the Widal serum reaction were to prove practiable.

Wagner has also very carefully studied the occurrence of genuine croupous pneumonia in enteric fever. In regard to the symptomatology he emphasizes, in the first place, in complete accord with Griesinger, that these pneumonias not very rarely are ushered in by a chill, accompanied by marked increase in the fever. Often there is marked or repeated recurrence of a flush upon the cheeks. The other signs are the usual ones of croupous pneumonia: dulness, bronchial breathing, etc. The course shows the same characteristic quick remissions after completed exudation as belong to the ordinary genuine pneumonia. Pneumonic sputum is almost always missed (not expectorated). Usually the pneumonia arises at the acme of the enteric fever, between the fourteenth and twentieth days; more frequently, perhaps, during the period of defervescence or even after convalescence has begun.

"Up to the present day, however, the existence of primary pneumotyphus is uncertain; that is, cases in which after several days of more or less characteristic symptoms of pneumonia the phenomena of an ordinary enteric fever arise, at one time with slight, at other times with decided development of the specific intestinal affection," says Wagner. He gives a complete review of the literature in relation to this subject. He declares, however, that it has not been possible for him, from the various, usually general descriptions of the disease, to gain an insight into the character of the real affection.

According to Liebermeister,* the pneumonias which arise at the acme of, or in the beginning of, convalescence from enteric fever present the type of the so-called secondary form. They usually manifest themselves by an increase in fever, occasionally by chill, or by the physical phenomena of infiltration. The cough is frequently scarcely increased; characteristic sputa are absent; pains are not present at all or, if present, unimportant. The same character is shown in the appearance of pneumonia in the first week of enteric fever.

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* Ziemssen's "Handbuch," 2d edition, 1876, p. 180.

Leichtenstern declares that the cases described under the name *pneumonia typhosa,* as well as those described first by Stoll and later by Traube and Mosler, as *pneumonia biliosa,* are identical with the form of "primary asthenic pneumonia" which he has described.

I concur with this opinion. Except in the sense of a pneumonia occurring during or after enteric fever, and demonstrated by the presence of an intestinal lesion, there can be no pneumotyphus. The variety characterized by this name belongs in the province of *atypical pneumonia,* which is to be described later on.

It must of course be remembered that not only genuine croupous pneumonia, but also *hypostatic pneumonia,* may occur in enteric fever. For the diagnosis of these two forms the fact may be used that the latter usually, although in unequal intensity, affects the two lower lobes; while the former also occur in the upper lobes.

Difficulties analogous to those present in pneumotyphus also develop in deciding the question whether in **intermittent fever** pneumonia occurs only as a simple complication of this disease, or whether there are pneumonic affections which represent a disease analogous with intermittent fever. Grisolle and Griesinger have treated this question explicitly. The latter says: "It seems that there are really rare cases in malarial districts that properly deserve the name *pneumonia intermittens;* in which chill, then fever, dyspnea, bloody sputa, crepitation in the lung, some dúlness upon percussion of the thorax, develop; where, however, with the perspiration and with the marked remission in the fever, also the objective symptoms markedly decline or even disappear. In a daily or tertian rhythm these attacks repeat themselves; with every paroxysm the infiltration becomes plainer, and also remains in the interval. After four or five paroxysms, as a rule, the patient dies." These cases are said to affect the left lower lobe exclusively. The process of infiltration follows rhythmically under the influence of the cause of the malarial fever; it may be compared to the affection of the spleen, in which the swelling at first becomes less in the period of apyrexia, and after several attacks remains permanently enlarged. The process can only be determined if it becomes possible to prove true intermissions, or very strong resemblances to such conditions in a simultaneous endemic or epidemic. The pneumonia of intermittent fever differs from those rare cases of ordinary pneumonia which are not caused by malaria, which are interrupted by remissions, and in which infiltration occurs, especially by the occurrence of chill, which always introduces the paroxysm of intermittent fever, but which is absent in the last-named cases.

[Pneumonia or a pneumococcus infection may set in in the course of malaria, but pneumonia due to the malarial parasite is unknown.—Ed.]

Finally, the development of pneumonia **during gestation** must be mentioned. This is an exceedingly dangerous complication. In the majority of cases pregnancy is interrupted, and the more advanced this may be, the more dangerous is the outcome. Grisolle gives a collection of 18 cases of pregnancy in which pneumonia occurred. Two of these already had valvular disease. Thirteen were not further advanced than the sixth month of their pregnancy; of these, 5 aborted upon the fourth, fifth, sixth, ninth, and fifteenth day of the pneumonia; three of these died a few hours later. The five women who were between the seventh and ninth months of their pregnancy all died.

According to the experimental investigation previously mentioned (p. 412),—which proved that in rabbits in which pure cultures of the bacteria of pneumonia were subcutaneously injected, abortion, followed by endometritis and peritonitis, occurred,—it may be assumed that the chief danger of pneumonia in pregnant women depends upon an abortion which is followed by a puerperal endometritis, which is produced by the pneumococci in the blood finding their way to the placental site in the uterus.

DURATION AND TERMINATION OF CROUPOUS PNEUMONIA.

The **duration** of croupous pneumonia may be from one day to three weeks; and in every case a complete *restitutio ad integrum* may occur. These extraordinarily great differences in the duration of the affection are nothing new for any one who has frequently had occasion to treat pneumonia; no author who has studied the laws of pulmonary inflammation would have any reason to object to this statement. Naturally, the number of the cases which terminate in the earliest and latest periods is a comparatively small one; perhaps the number of such cases which terminate in the first two or three days by crisis would be greater than is usually supposed if there were not many physicians who are very skeptical about the diagnosis of a "one- or two- or three-day pneumonia" because they have had the opportunity of observing the disease from the first day and the objective findings are not positive.

The number of one- to two-day pneumonias which have been determined with certainty is therefore small; their existence, however, cannot be questioned. Thomas has described three cases of a two-day duration. Tophoff, Baruch, and Bernhard have each described a case of a similar duration. Leube describes two cases of pneumonia lasting one day. Weil had an opportunity to observe such a one-day pneumonia from the first moment of its development, because from the onset of the affection the patient was present in the hospital.

The pneumonia ran its course with all the characteristic symptoms, but even eight days later crepitation was still to be heard over the diseased left lower lobe. In this connection Weil gave the opinion that such pneumonias with one-day fever may occur more frequently than is generally supposed; many a *status febrilis*, many a *febris ephemera*, whose cause cannot easily be found may etiologically belong in this group of pneumonias. He advises also, after the defervescence, at a time in which the general condition of the patient is favorable, to examine the lungs very carefully. An increase in the respiratory frequency, with slight dulness, a few râles, or the sputum may occasionally give the proper diagnosis, and prevent the physician from making the diagnosis of *febris ephemera* of which he is frequently ashamed. [We have seen one case of two-day pneumonia with all the characteristic symptoms and signs. Bronchial breathing continued for four days after the crisis.—Ed.]

Wunderlich counts pneumonia of brief duration among the abortive variety. Finkler calls them rudimentary pneumonias. [Broadbent reports a case with abortive crisis and premature resolution in forty-eight hours. The toxemic symptoms were very severe.—Ed.]

The question as to the character of the local changes which are unquestionably present has never been decided. Above all, it is certain that such pneumonias do not run through all the anatomic stages; even the development of red hepatization is not conceivable, because, according to universal experience, this cannot be completely developed before the third day.

An explanation of the anatomic process in rudimentary pneumonia was not possible so long as the first stage of pneumonia, engorgement, was supposed to be due to a hyperemia of the alveolar capillaries, with a subsequent exudation of white blood-corpuscles into the alveoli. At least no one would like to decide that the hyperemia alone could be looked upon as the cause of the disease, and that with its cessation the disease terminates; an emigration of white blood-corpuscles at the beginning of the disease would have to leave traces which could not disappear so rapidly. The correct interpretation of the process must be based on the fact, determined by anatomic investigation, that the affection of the alveolar epithelium represents the beginning of the disease, and only after this has occurred a capillary hyperemia takes place. During the stage in the alterations of the alveolar epithelium depicted in Plate 4, figure 1—that is, the stage of swelling of the epithelium, possibly with the escape of red blood-corpuscles into the alveoli—the process may undergo regeneration within a few days, if the cause,—that is, Diplococcus pneumoniæ,—from any circumstances unknown to us, loses its virulence.

The other extreme, excessive duration of the pneumonia, or at least of the fever, may be due to two different causes. Either resolution in the disease, which from the beginning was localized, is delayed; or other portions of the lung are attacked in addition to the part originally involved.

In the former case, when the inflammation remains limited to the originally diseased lobe, the excessive duration can be tentatively explained on the supposition that the inflammation has extended locally in the form of an abscess, or has diffused itself in the interstitial tissue, without, however, completely excluding the possibility of recovery. The defervescence under these circumstances is usually by lysis.

The second cause, the subsequent involvement of other parts of the lung, before the first part affected has run through the cycle of the anatomic stages, is the more frequent. Such terminations of pulmonary pneumonia are usually designated **wandering pneumonia,** but with this no exact definition is given. It certainly does occur that, for example, through an inflammation of the right lower lobe, in the further course of the affection, one of the left side of the lung may arise. Here no wandering of the inflammation can have occurred; the causes of the disease must be independent local ones. Yes, the question might even be asked whether in the later affection of a lobe adjacent to the primarily diseased one, the conditions for the later affection were not originally present in this lobe, and the supposition of a slow advance of the inflammation is far less entitled to consideration than this supposition. It at least rendered a uniform conception possible, whereas the supposition of a wandering pneumonia is not applicable to a later affection of a lung, which up to that time had remained intact.

At all events, as the proof is wanting for the correctness of the proposed question, it is more correct not to designate the process a *pneumonia migrans,* as too much is presupposed with this, but to designate it a pneumonia with relapses.

An explanation of these relapses can only be purely hypothetic. It must be based upon the foundation that the process which has led to an inflammation of the lung has only been active during a certain time. If this process, however, at the onset has only been active at one place, and only later becomes manifest in another position, this can only be ascribed, in consideration of the predisposition existing in the latter place, to the fact that Diplococcus pneumoniæ has been delayed in developing, in consequence of the influence of the products of metabolism which were introduced into the blood from the originally diseased area. I am led to this assumption by some observations upon the retardation of the action of specific bacteria present in the human organism by the simultaneous presence of other organisms of greater virulence. I have several times seen that a gonorrhea, which was acquired before the development of enteric fever, only became noticeable after the cessation of the former process.

From the pneumonia with relapses which has just been mentioned **recurring pneumonia** is to be differentiated. This has given rise to many explanations which have had for their object the description of an exact differentiation. Ruge has given very explicit statements in reference to this. I, for my part, think that the definition given by Wagner is the best. He says: "If the lung, after an ordinary croupous pneumonia involving one or several lobes, becomes normal after the fever has terminated by crisis or lysis, the patient is convalescent; and if at least three days to several weeks after defervescence a new infiltration of the same or other lobes with all the characteristic phenomena of a local and general nature occur, a relapse has without question taken place." Recurring pneumonia is undoubtedly exceedingly rare. According to Grisolle, it usually takes place between the thirteenth and sixteenth days of the disease. [We have seen a relapse at the end of five days.—Ed.]

A more prolonged course, but one which nevertheless may terminate in recovery, is **delayed resolution.** Leyden has described this minutely. He says that in old, debilitated, or cachectic individuals, or in patients weakened by sequels, occasionally some weeks may elapse before the infiltrated parenchyma of the lung again takes up air. In such a case it is only a question of weeks, and resolution proceeds hand in hand with the other phenomena of convalescence. There are, however, cases in which the pneumonia runs a typical course and terminates by crisis, but in which no resolution occurs; although the patient is more or less robust and gains strength, the infiltration nevertheless remains, not for weeks, but for months. The pneumonia therefore terminates in permanent infiltration or induration, and the fear arises that a *restitutio ad integrum* will not occur at all, and that a chronic consolidation or a caseous pneumonia will develop which may lead to phthisis. [Tuberculosis does not follow croupous pneumonia. —Ed.] If one follows such cases, it will be noted that they retain the physical signs of an infiltrated lung that is on the point of undergoing resolution without or with only slight modification; bronchial breathing and crepitant râles at the height of inspiration remain; never are there moist râles of large caliber or any signs of degeneration of the tissue or pus formation, and no retraction that can be noted occurs. The general condition is satisfactory. The patient gains strength. Upon careful comparison one notes, however, no matter how slowly, a gradual clearing up of the percussion note, and in some few isolated areas the return of the respiratory murmur to normal. The physical signs retain the type of a very gradual resolution, and this decides the question. The sputum also retains the properties belonging to the stage of resolution of pneumonia. It is scanty, greenish-yellow, and consists of small plugs or balls of pus-corpuscles and alveolar epithelium which has undergone fatty degeneration.

The termination in **chronic pneumonia** [in abscess and in gangrene. —Ed.] will be spoken of later, in a section devoted to that purpose.

More frequently than this is the production of diffuse **bronchiectasis** as the result of pneumonia. The occurrence of this sequel has been described by me elsewhere, and by Hoffmann in another part of this collective work.

MORTALITY AND DIRECT CAUSES OF DEATH.

The mortality of croupous pneumonia is due to a great number of causes, an analysis of which is indispensable if appropriate means are to be devised to prevent the action of the individual factors. [Wells, in an analysis of 223,730 cases collected from various sources, found a mortality of 18.1%. The hospital mortality is high—ranging from 20% to 40%. At the Presbyterian Hospital, Philadelphia, the mortality was 28.7% in 195 cases; Montreal General Hospital, 20.4% in 1012 cases; Charity Hospital, New Orleans, 38.01% in 3969 cases; Johns Hopkins Hospital, 25% in whites, 30% in colored; Pennsylvania Hospital, Philadelphia, 29% in 704 cases; Boston City, 29.1% in 1443 cases. Townsend and Coolidge show by an analysis of 1000 cases that the mortality in the Massachusetts General Hospital was a little over 10%, and that the death-rate has not increased in recent years. In private practice Fussel had a mortality of 17.9% in 134 cases; and R. P. Howard (quoted by Osler), of 6% in 170 cases.—Ed.] One cause is certainly predominant which is not dependent upon locality, upon season, or upon the individual. But its presence can only be determined because the three chief conditions which have just been named are not sufficient to explain a fact that is undeniably true, and is brought out by an analysis of the causes of mortality. This fact is that the mortality of pneumonia varies greatly in *different years*. In one and the same city, in one and the same hospital or district, in persons who in the main do not show the slightest difference in their living conditions in the course of years, when the pneumonia is treated by one and the same physician in the same manner, the mortality may in the course of these years be an astonishingly unequal one. Brandes has already called attention to the enormous differences in the mortality in separate years, although only to criticize the methods of treatment in vogue. He says verbatim: "If one obtains in a disease by the use of the same method, in one year a mortality of 5%, in the next year a mortality of 31%, this shows that the therapeutist cannot rely on statistics based on an enormous number of compiled cases." So enormously great the differences certainly are not. If one deduct the cases complicated by delirium tremens, then for the most unfavorable year there remains a mortality of but 26%. Nevertheless the difference is a decided one.

In the 2626 cases of pneumonia treated during the years from 1840 to 1855 in the statistics of Huss, the mortality was:

In 1851 .... among 242 patients 18; or 7.43%.
" 1843 .... " 134 " 19; " 14.17%.

The maximum is therefore almost twice as large as the minimum. "The only conclusion which may be drawn from these differences is that the pulmonary inflammations which occurred in one year were more severe than those which occurred in other years."

According to Fraenkel's and Reiche's reports, there died in the cases treated in the Hamburg Hospital:

In 1889. .men, 36 = 19.0%; women, 2 = 5.3%; together, 38 = 16.7%.
" 1890. . " 43 = 15.9%; " 15 = 27.8%; " 58 = 17.85%.
" 1891. . " 51 = 14.2%; " 26 = 29.2%; " 17 = 19.1%.
" 1892. . " 49 = 24.0%; " 12 = 20.7%; " 61 = 23.3%.

Very clearly my own observations illustrate the fact of the very variable mortality in special years.

THERE WERE TREATED IN:	MEN.	WOMEN.	TOGETHER.	OF THESE THERE DIED:			
				Men.	Women.	Together.	Therefore in percentage:
1880	74	7	81	8	–	8	9.8
1881	74	14	88	7	3	10	11.3
1882	67	13	80	13	1	14	17.5
1883	75	17	92	17	3	20	21.7
1884	66	10	76	6	3	9	12.0
1885–86 *	65	14	79	20	5	25	25.3
1886–87	101	15	116	20	2	22	19.0
1887–88	126	12	138	26	2	28	20.3
1888–89	63	18	81	13	3	16	19.7
1889–90	83	15	98	17	5	22	24.4
1890–91	123	25	148	15	4	19	13.0
1891–92	86	17	103	13	5	18	17.14
1892–93	118	31	149	14	4	18	12.1
1893–94	35	28	63	4	4	8	12.6
1894–95	34	15	49	7	5	12	24.9
1895–96	33	27	60	2	2	4	6.6

The periodic differences become even more conspicuous if I add that, especially in the first years, a completely neutral therapy was used—no alcohol, no quinin, no baths; and with this the most favorable mortality. But the courage to continue this therapy weakened from year to year. The highest mortality cannot be altogether explained by the excessive use of alcohol in the form of Hungarian wine, to which I was forced, against my will, by the pressure of professional opinion, as well as the firm belief of my assistants, who had come from various universities. I would have been accused of the sin of omission had I done otherwise. Only after the experience of the years from 1886 to 1888 I limited the use of stimulants. Under these circumstances nothing else remained for me than by the method of exclusion of all local, seasonal, individual therapeutic influences to come to the conclusion, which was unquestioned by me, that the variation in mortality in the separate years was produced

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* From this time on, the enumeration occurred according to the fiscal year,—that is, from the 1st of April to the 31st of March,—whereas up to that year the calendar year was used.

by the difference in the strength of the virulence of the pneumococcus itself. Such an assumption can of course receive complete confirmation only by a positive examination of a periodic difference in the virulence of every special kind of pathogenic bacteria, which up to this time has not yet been possible. Clinical observation allows us, however, to expect such a confirmation with confidence.

Of great importance next to this in influencing the mortality is **age.** In reference to this there exist many statistical reports, but many of them are entirely worthless. Either they have been collected from the civil population and do not rest exclusively upon reports by physicians, or they refer to hospitals into which children and certain other classes of individuals are not admitted. Even the figures of so careful an observer in pulmonary inflammations as Huss do not allow of positive deductions. Among 2616 pneumonia patients, children up to the fifth year of life have not been noted; between the fifth and tenth years only 9 were noted; there were, however, between the ages of twenty and thirty 1040. The report that of the 9 children 1 died—11.1%—cannot be taken as conclusive.

The statistics of Fraenkel and Reiche in reference to age are of especial value. In their 1130 cases death occurred at the following ages:

1–5	years	9	times in	30	cases	= 30.0%
6–10	"	2	"	52	"	= 3.84%
11–20	"	11	"	219	"	= 5.0%
21–30	"	31	"	355	"	= 8.7%
31–40	"	57	"	231	"	= 24.7%
41–50	"	53	"	135	"	= 39.3%
51–60	"	28	"	65	"	= 43.1%
61–70	"	15	"	28	"	= 53.6%
71–80	"	13	"	15	"	= 86.7%

SHOWING DEATH-RATE OF PNEUMONIA AT VARIOUS AGES—PER CENT.—(*Wells.*)

AUTHORITY.	—10	10–20	20–30	30–40	40–50	50–60	60–70	70+
Bamberger	..	4.0	3.2	5.2	7.0	35.0	40.0	50.0
Biach	..	9.4	13.3	23.3	31.6	37.8	54.0	56.6
Chomel	..	..	13.6	18.0	33.3	40.0	63.0	..
Derpmann	26.3	49.2	48.9	38.9	48.1	40.6	11.5	21.2
Doubleday	..	21.5	33.3	53.3	70.7	66.6	56.2	50.0
Franque	..	3.1	10.9	23.5	33.3	76.9	..	..
Huss	11.1	7.8	7.9	15.1	23.7	26.9	38.9	60.0
Jürgensen	3.0	..	4.0	6.0	10.0	12.0	16.0	11.0
Klinger	43.4	2.2	4.3	5.0	7.3	12.2	17.8	10.7
Lépine	..	..	14.0	20.0	25.0	30.0	30.0	80.0
Pause	26.5	..	..	17.2	10.5	11.1	35.7	38.4
Roth	..	4.0	7.6	20.9	26.7	32.3	26.7	60.0
Schapira	..	1.4	2.8	11.4	21.7	24.0	50.0	75.0
Stechert	46.1	6.8	7.2	21.1	40.7	52.0	66.6	66.6
Stortz	..	2.4	6.0	10.5	26.8	33.3	50.0	50.0
Swett	..	..	24.4	14.3	16.6	20.0	25.0	..
Townsend and Coolidge	10.0	10.6	18.5	25.3	29.2	55.0	46.9	66.7
Wells	8.0	3.1	7.0	9.4	13.0	24.0	37.0	53.0

These tables in the most important points coincide with my findings:

The Total Number According to Age.	Men.	Women.	Total.	Fatal Cases.			
				Men.	Women.	Total.	Percentage.
Up to 5 years	35	22	57	11	3	14	24.4
" 10 "	22	24	46	0	0	0	0.0
" 20 "	256	77	333	5	6	11	3.3
" 30 "	372	65	437	30	9	39	9.0
" 40 "	229	30	259	40	7	47	18.1
" 50 "	172	23	195	48	5	53	37.0
" 60 "	69	14	83	30	7	37	44.5
Over 60 "	68	23	91	38	14	52	57.0

It is conclusively proved by both these tables that the mortality steadily increases from the sixth year to old age. Conspicuously favorable is the outcome of the affection between the fifth and tenth years of life, whereas under the fifth year the prognosis is extremely unfavorable. I was disposed to assume that in former years some cases that terminated fatally up to the fifth year of life, and upon which I held an autopsy, were cases of catarrhal pneumonia, and that I regarded them as cases of croupous pneumonia on account of their lobar extension, but the conspicuous coincidence of my reports with those of Fraenkel and Reiche entitles me to drop this suspicion.

Several reasons have contributed to the contradiction existing in the views in reference to the danger or harmlessness of pneumonia in **childhood.** What value can numbers have in reference to the prognosis of pneumonia in children if the statistics include all cases up to the twentieth (!) year of life, whereas, to be of value, at least the first lustrum of life should be separated from the second, or even each year up to the tenth should be considered by itself. Thomas has added up the reports of twelve authors, all of whom showed very favorable results. Three of these counted up all their cases up to the tenth year of life, three up to the fifteenth, three up to the twentieth year, and upon this built their conclusions as to the favorable course of the affection in childhood. In three of the twelve authors mentioned exact reports are missing altogether.

My reasons for refusing in spite of this to accept the above statistical reports in reference to the mortality up to the fifth year of life as generally applicable are based on my observations in private practice. Unfortunately I have no exact notes on which to base a statistical report. I can only say that in my private practice I have had much more favorable results in children of the previously mentioned age. I am under the impression that the prognosis is more favorable in children treated in the hospital, on account of previous unfavorable conditions in reference to nutrition.

Great influence upon the mortality is ascribed to the **sex** of the patient. Grisolle[519] maintains, with Briquet, that the mortality in women is doubly as great as in men. He ascribes this fact partly to the greater average age of the affected women. An analogous condition as to mortality in women is seen from the reports of Geissler.

Of 347 men, 62· died; or 17.9%.
Of 78 women, 25 died; or 32.5%.

The figures of Huss likewise speak for a greater mortality among females, although not in such a high degree:

Of 2187 men, 219 died; therefore 10.01%.
Of 429 women, 62 died; therefore 14.45%.

My own figures force me to the opinion that the mortality among females is not greater than among males. In reference to this point statistics may be regarded as absolutely conclusive if, as in my case, they are based on a large number of cases, and the mortality in the female sex is not found to be higher than in males. This must then be sufficient to prove that there must be other reasons than sex in producing differences in mortality.

Of 1501 cases of pneumonia,	253 died;	or 16.8%.
Of 1223 men,	202 died;	or 16.5%.
Of 278 women,	51 died;	or 18.0%.

These figures receive valuable support from the comprehensive statistical investigation of v. Ziemssen. According to his count, which includes the statistical reports of different countries from 1839 to 1856, the percentage of females in the mortality of pneumonia was:

In Paris.	In Copenhagen.	In Hamburg.	In Zurich.	In Berlin.
48.1%	41.1%	43.4%	51.1%	43.9%

TABLE SHOWING COMPARATIVE MORTALITY OF PNEUMONIA BY SEXES.—(*Wells.*)*

Observer.	Males.			Females.			Total.		
	Cases.	Deaths.	%	Cases.	Deaths.	%	Cases.	Deaths.	%
Berlin Charité	453	90	20.0	114	26	23.0	567	116	20.5
Biach	8,247	1,707	20.7	3,195	959	30.0	11,442	2,666	24.2
Chomel	313	38	12.5	70	22	32.0	383	60	15.6
Dietl	364	28	6.7	297	41	12.1	661	69	10.4
Huss	2,259	291	12.9	451	84	18.6	2,710	375	13.8
Huss, Vienna cases	3,981	831	20.9	2,009	610	30.4	5,990	1,441	24.5
Jürgensen	5,467	1,149	21.0	2,475	795	31.1	7,942	1,944	24.9
Louis	52	15	29.0	23	3	13.3	75	18	24.0
Munich Hospital	794	132	16.6	352	81	23.0	1,146	213	18.5
Pause	86	14	16.2	83	12	14.4	169	26	15.3
Stecher	548	95	17.3	108	33	30.6	656	128	19.5
Stortz	199	23	10.6	87	22	25.3	286	45	25.7
Townsend and Coolidge	724	182	25.0	276	68	25.0	1,000	250	25.0
Waller	71	7	10.0	10	2	20.0	81	9	11.1
Wells	295	41	14.3	203	40	19.7	498	81	16.4
Totals and averages	23,853	4,643	19.4	9,753	2,798	28.7	33,606	7,441	22.1

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* "On Mortality of Pneumonia," Wells, *Jour. Amer. Med. Assoc.*, 1890.

If we examine the local conditions that influence the outcome of the disease, one of the first points to be taken into consideration is the **location** of the pneumonia.. According to Auenbrugger and Corvisart, left-sided pneumonias are more unfavorable than right-sided ones. Briquet believes that the contrary is true. Grisolle has noted no differences in the two sides in reference to the influence upon the mortality.

We are no better off in deciding the question whether pneumonias of the upper lobes are more dangerous than those in other parts of the lung. Andral and Grisolle believe that the former are more dangerous; Briquet does not believe that they have any especial influence upon the mortality.

In regard to the two questions, the prognostic influence of the right side as compared with the left, and the upper as compared with the lower lobes, a tabular arrangement of my 253 deaths shows the following distribution of the pneumonic lesion:

LOBE.	MEN.	WOMEN.	TOTAL.	
R. L.	33	6	39	120 = 47.0%.
R. U.	15	4	19	
R. 2 lobes	19	5	24	
R. lung entire	32	6	38	
L. Lower	34	3	37	68 = 26.8%.
L. U.	6	1	7	
L. lung entire	17	7	24	
Both lungs	46	19	65	65 = 26.2%.
Total	202	51	253	

In the 19 cases in which only two lobes upon the right side were diseased the inflammation was distributed as follows:

R. 2 LOBES.	MEN.	WOMEN.	TOTAL.
R. U. plus middle	13	2	15
R. L. plus middle	4	3	7
R. U. plus R. L.	2	0	2
Total	19	5	24

The right upper lobe was affected in 19 cases = 7.5%.
The left upper lobe was affected in 7 cases = 2.8%.
Total, ... 26 cases = 10.3%.

Analysis of the fatal cases alone shows a decided preponderance in favor of the right side, but the difference appears very much smaller when the number of right-sided pneumonias is compared with the total number of cases. Whereas in 189 cases the inflammation was present upon both sides, the right side was affected 782 times and the left 570.

In 782 right-sided pneumonias there were 120 deaths = 15.3%.
In 530 left-sided " " " 68 " = 12.8%.

From this it would appear that right-sided affections are in themselves more dangerous.

The upper lobe alone was attacked:

145 times upon the right side with 19 fatal cases; or 14.0%;
57 " left-sided, " 7 fatal cases; or 12.3%;

giving such a slight difference that no importance can be attached to this, especially in view of the comparatively small number of cases.

Of unquestioned influence upon the mortality are some affections which have been present prior to the pneumonia.

As may be inferred from previous remarks in reference to affections which may have been present prior to the occurrence of pneumonia, *chronic nephritis* and *cirrhosis of the liver* almost invariably have a very unfavorable influence upon the prognosis. *Valvular disease* of the heart is exceedingly dangerous. It does not, however, exclude a favorable outcome. The same is true of *scoliosis*. *Pregnancy* is one of the most dangerous complications; the danger is the greater the nearer the patient is to the normal termination of pregnancy. The contractions of the uterus which lead to abortion or to premature birth usually occur upon the third or fourth day of the disease (Klautsch). *Delirium tremens* also increases the mortality. It is, however, more favorably influenced by proper therapeutic measures than any other complication.

The prognosis of *contusion* pneumonia has been given variously. According to Litten, it runs a course similar to that of an ordinary spontaneous attack and does not give an unfavorable prognosis. Paterson, on the other hand, in five cases of traumatic pneumonia had three deaths.

In addition to these conditions which influence the mortality from the beginning, there are certain symptoms produced by the pneumonic affection itself which affect the prognosis.

A *prodromal period* lasting several days, combined with nausea, makes the prognosis questionable (Grisolle).

High fever, especially in the morning, if it amounts to 40° C. (104° F.) and lasts more than seven days, is serious.

A *pulse-rate* greater than 120 per minute is unfavorable.

Severe *bronchial catarrh*, and, as an accompaniment of this condition, marked cough which disturbs the night's rest, with severe pains in the side, have an unfavorable influence upon the outcome of the affection if the symptoms cannot be checked; fortunately it is usually possible to do so.

The occurrence of *herpes* in the course of pneumonia is said to be favorable; the exact investigations of Geissler seem at first sight to substantiate this opinion. His analysis shows that:

Of 421 patients	87, or 20.70%, died.
Of 182 with herpes	17, or 9.30%, died.
Of 239 without herpes	70, or 29.30%, died.

The result, however, is different if one considers the occurrence of herpes in men and women:

Of 156 men with herpes	10, or 6.45%, died.
Of 27 women with herpes	7, or 25.00%, died.

Further, from the report of Geissler, the great frequency of herpes occurring in the first period of life is seen (50% of herpes in childhood, as opposed to 43% between the ages of fifteen and thirty, in his total of 421 cases). In childhood, however, he had a very slight mortality—6.54%.

We must conclude that herpes in itself cannot be looked upon as a favorable sign in prognosis and that it occurs mostly in childhood, when the prognosis in itself in pneumonia is more favorable.

To make use of the condition of the blood, especially *leucocytosis*, as a prognostic sign, is not possible. [It is now believed a *leucopenia* is an indication of a very grave case. (See Leucocytosis.)—Ed.] Although the number of the white blood-corpuscles in comparison to the red rises with the increase in the fever, Halla, Rieder, v. Jaksch [and Osler.—Ed.], have especially noted in the cases which terminate fatally a decided diminution of the leucocytes.*

The occurrence of *pneumococci* in the blood, according to Kohn, is to be looked upon as an unfavorable prognostic omen. Among 32 cases of pneumonia he found pneumococci 9 times by means of puncturing a vein of the arm. Of these 9, 7 died; 2 recovered, however, one after a pyemia with metastatic abscesses, and the other after an empyema.

Finally, the fact which is readily understood, that danger to life increases with the extent of pneumonic consolidation, must be mentioned. It might be added that this is, strange to relate, not true to the extent one might suppose *à priori*. Recoveries from pneumonia of an entire lung, and even a lobe of the other lung, do not belong to the curiosities of this affection. The fact mentioned above, that in 3 fatal cases both lungs were entirely consolidated,—only a part of the upper lobe contained air,—proves how long life may exist. Naturally, in cases that run a favorable course it must always be supposed that some parts of the lung which, according to the clinical examination, are supposed to be completely devoid of air, have still been able to carry on an interchange of gases.

If, however, the fatal outcome is to be referred to the lungs themselves,—which in a part of the cases is undoubtedly correct,—it is due in the majority of cases to the *edema* which affects those parts of the lung that are not implicated in the pneumonic infiltration. I pass over the earlier opinions in reference to the production of edema of the lungs, because they have little value. I shall only mention the investigations of Rivalta, which would have especial value if they were confirmed by other investigators. Of 52 cases terminating fatally, in which an autopsy was performed, edema of the lungs occurred 33 times, or in 65% of the cases; 22 times the edema was present in the entire lung; 6 times at the base of one lung; twice at the base of both lungs, and three times in an upper lobe. In the edematous parts Rivalta constantly found Fraenkel's diplococcus in large numbers, and almost exclusively in pure culture. In addition, a number of the alveoli presented a condition of acute edema analogous to a beginning exudation, which was due to the mixture of red and white blood-corpuscles with desquamated epithelium. Upon the basis of this Rivalta declares that the edema in pulmonary inflammation is due to the diplococci, which cause an *inflammatory acute edema.*

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* See case of relapsing lobar pneumonia with absence of leucocytes, Stockton, *Phila. Med. Jour.*, 1898.

The *causa proxima* of the fatal outcome in an overwhelming proportion of cases is the gradual loss of power in the heart. "Patients who die of pneumonia are killed by cardiac insufficiency," says v. Jürgensen. This assumption he bases upon the following facts: (1) The pneumonic exudation increases the resistance in the lesser circulation and in this way throws an increased amount of work on the right ventricle; (2) the changes produced by pneumonia at and in the lung produce a diminution of the movement of the blood through these organs, especially as a part of the normal power of the circulation disappears, and thus prevents the expansion of the sound lung, whereas just those parts of the surface of the lung which have been diminished by the pneumonic exudate require greater power to force on the blood for the necessary interchange of gases to occur.

"The fever, however, brings the disturbances locally produced by the pneumonia to the surface. It increases the action of the heart, while at the same time it directly injures the viscus," v. Jürgensen adds.

Now, the statement that the final outcome of pneumonia is to be attributed to insufficiency of the heart is unquestionably correct, but the assumption that the fever is the cause of the cardiac insufficiency cannot be taken as final. Against this the following facts must be noted:

In the first place, the degeneration of the muscle of the heart—which v. Jürgensen, with Zenker and Liebermeister, looks upon as the result of the fever—need not at all be conspicuous, even if death in pneumonia has occurred under symptoms of cardiac insufficiency. Further, death may have occurred with the phenomena of cardiac insufficiency, even if the fever has not been conspicuously high. Just so in other diseases, a very decided degeneration of the heart muscle may occur with slight or even absence of fever. Finally, other diseases may be characterized by greater fever of much longer duration,—for example, enteric fever,—and still do not give rise to the assumption that cardiac insufficiency is as frequent a cause of death as it is in the case of pneumonia.

Lately Bollinger has given expression to the view that the collapse phenomena occurring at the time of crisis in croupous pneumonia, and the lethal cardiac insufficiency, are particularly due to oligemia, which leads to insufficient nourishment of the already febrile weakened heart; the simultaneous anemia of the brain may also give rise to disturbances of innervation of the heart muscle which have an unfavorable influence.

In reference to the production of oligemia in pneumonia with a plentiful exudate, he declares, upon the basis of pathologic-anatomic investigations, that the former is due to the vascular inflammatory exudate which develops in the course of a few days similar to an internal hemorrhage. The action of the pneumococci upon the pulmonary exudate may be likened to venesection, which in a few days may take from the blood large quantities of important constituents. That the fatal outcome of pneumonia occurs so comparatively early, and almost always in the same stage (between the sixth and eighth days, corresponding to the transition period from red to gray hepatization), is probably due to the fact that the exudate must reach a certain maximum, with which life is no longer compatible; that at the same time the fever, and the infection and intoxication which accompany it, are important prognostic factors, is readily understood.

In answer to this view it is to be observed that the explanation of cardiac insufficiency by the escape of blood into the alveoli of the lung, as in copious venesection, is not compatible with clinical observation. In pneumonia there is no symptom comparable to the signs of blood loss in other febrile affections; for example, with acute hemorrhage from ulcers of the bowel in enteric fever.

According to my opinion, the unfavorable influence upon the heart, as in many other severe febrile infectious diseases, is due to the action of poisons produced by bacteria which reach the heart muscle through the blood stream. [The toxemia.—Ed.] The uniformly injurious effect upon the heart of various infectious diseases forces me to this explanation. That the effect varies much in intensity is due to the condition of the heart muscle itself. Previous conditions of weakness of the myocardium influence the outcome of the disease in an especially high degree.

[In fact the cause of death in pneumonia in healthy subjects is usually either the simultaneous infection of other organs, as the meninges, the endocardium and pericardium, the pleura, and other structures with the pneumococcus; or a toxemia, the degree of which depends not alone upon the extent of lung involved or of other structures invaded, but upon the virility of the infection and the condition of the patient. A *pneumococcus septicemia* is induced. This toxemia is indicated by nervous symptoms, as in the typhoid state, by progressive cardiac weakness, and by diarrhea and meteorism. Heart failure may be induced by this toxemia or by overdistention of the right heart. Death from mechanical interference with respiration is very rare.—Ed.]

DIAGNOSIS OF PNEUMONIA.

A number of other affections may render the diagnosis doubtful, depending on the time of the appearance of the individual symptoms in the course of the disease.

If the pneumonia develop gradually, and only an indistinct sensation of malaise precedes, there can of course be no question of a diagnosis. Even if some cough should occur, the situation would not be especially cleared up. The fact that bodily exertion or the influence of unfavorable weather conditions have preceded, or both together, requires notice; but in this connection it is to be remembered that under these circumstances, just as well as without any preceding injury, a general feeling of malaise combined with cough, pointing to a disease of the lung, need not necessarily be a croupous pneumonia, but it may also be the beginning of an acute [tuberculous.—Ed.] infiltration of the apices of the lung or even of a *miliary tuberculosis*. In all these conditions there is slight fever, frequently with chilliness, and general sensations of malaise. In miliary tuberculosis of the lung, as in croupous pneumonia, the fever may show acute decided rises, and both the morning and the evening temperatures may be persistently high, with only a slight morning remission. In favor of croupous pneumonia would be the onset with a decided chill, introducing the rise in temperature, but only the subsequent course, especially the appearance of characteristic phenomena over the bases, would give certainty to the diagnosis. Should, however, the disease arise in an upper lobe, the most characteristic symptoms, such as rusty sputum, marked dulness, and bronchial breathing, may be absent and make the diagnosis all the more difficult. If we have to do, then, with an acute miliary tuberculosis, the ordinary very stormy course with conspicuous inconstancy and incongruity of the objective findings over various parts of the lung are of especial importance; the further course of the affection brings the sad explanation. But, on the contrary, a pneumonia of the upper lobe beginning gradually, with slight irregular fever and absence of pneumonic sputum, may be mistaken for a tubercular infiltration. I have, in the course of my hospital practice, noted several cases in which the diagnosis of "tubercular infiltration of the apices of the lung" had to be made, in spite of the absence of tubercle bacilli. Only in the last years was it possible for me in a few cases of this kind to make a provisional diagnosis of "croupous pneumonia of the upper lobe," and to find it confirmed by the fact that the patients were discharged cured and able to carry on their work. [Elastic tissue and tubercle bacilli in the sputum will decide the diagnosis, although they may not be found until late in tuberculo-pneumonic phthisis.—Ed.] If anywhere, then here in these processes, which are so exceedingly difficult to differentiate, it is necessary for the diagnosis to lay stress upon the history of the patient, and not to neglect this for objective methods of investigation. A good history is half the diagnosis, occasionally the entire diagnosis.

[Recent studies show that valuable aid in the diagnosis of pneumonia can be obtained by a study of the leucocytes, by the diaphragm phenomena, by the use of the x-rays, and by the finding of pneumococci in the blood or inflammatory products. An absence of leucocytosis is very strong negative evidence that pneumonia does not exist, unless in the very mild form or in the opposite condition where there is extreme toxemia. Cabot states that in acute tuberculous pneumonia an increase of white cells may be found, although not invariably so. The x-rays indicate the presence of consolidation by the darkened lung, the restriction of the movements of the diaphragm, and by the displacement of the heart.

The diaphragm phenomena enable one to distinguish between consolidation of the lungs and affections of the liver and subdiaphragmatic abscess.—Ed.]

If *vomiting* usher in the pneumonia, which, on the whole, is rare without chill, the diagnosis cannot longer remain in doubt. Although in other acute affections, especially in scarlet fever, in the various forms of meningitis [in appendicitis.—Ed.], vomiting may open the scene, and fever, as in pneumonia, immediately follow the vomiting, and also increase rapidly to the same degree, the objective phenomena in such cases, even if they do not begin with a stitch in the side, appear inside of the first twenty-four hours, and are sufficiently plain to make certain of the case if one does not neglect to carefully examine the lungs.

That *convulsions* in children may represent the onset of pneumonia is a fact which must be remembered. The frequency of these convulsions is slight compared to convulsions due to other causes, but the acuteness of the physician who bears this connection in mind shows itself here in its best light. If no other disease, especially an *acute gastro-intestinal catarrh*, which occasionally may give rise to fatal convulsions, or *inflammation of the kidneys*, has preceded, it is necessary, in the first place, to determine in these convulsions, which arise in apparent complete health, whether *fever* is present or not. If this be the case, it must be determined whether *paralysis* has followed these convulsions, and then only the possibility of the origin of a pneumonia must be thought of, a determination of which depends upon repeated examinations of the lung. I have, in fact, once, in connection with convulsions which were followed by decided fever, seen a purulent panophthalmitis of the eye in a child aged one year.

For the acute chill which apparently arises without cause, croupous pneumonia is the most likely condition. If a stitch in the side accompany or precede this, there exists in previously healthy persons the greatest likelihood that we have to do with the onset of a croupous pneumonia; absolutely certain the diagnosis is not, even here. A beginning *mastitis* may—with insufficient objective examination—apparently begin similarly.

If, however, a glassy mucoid expectoration and rusty sputum occur, the condition is very much more certain; but even then the possibility of a confusion with a well-developed formation of infarct in the lung, as the result of local thrombosis or as the result of embolism, is not excluded. The presence of thromboses in the extremities or in the peri-uterine veins (during the puerperal period) and the existence of valvular disease of the heart which may give occasion for the production of infarcts are in such a case of importance for the diagnosis. The sputa in such cases are almost exclusively bloody.

In regard to the symptoms that are determined by examining the lungs, there are some diagnostic factors that deserve attention. The crepitant râles characteristic of the initial stage of pneumonia may also occur in chronic infiltration of the upper lobes, and may represent the only demonstrable auscultatory sign in cases that are not very far advanced. The sign has also been observed in pulmonary infarction; and although Grisolle asserts that the crepitant râles of pneumonia differ from the crepitation heard in pulmonary edema or hemorrhagic infarct, and theoretically a difference no doubt exists, an attempt to differentiate between the two varieties of crepitation in practice appears risky. Although it is undoubtedly true that in pulmonary edema, as in hemorrhagic infarction, the bubbles produced by the mixture of tenacious mucus with air are made in bronchi of larger caliber than is the case in the initial stage of pneumonia, so that the sound produced by the bursting of the bubbles—in other words, the crepitant râle—is higher in pitch in pneumonia, nevertheless this theoretic reasoning about the nature of a crepitant râle may in practice be absolutely worthless if there happens to be catarrh of the finer bronchi during the initial stage of pneumonia. If a purely physical explanation of the objective symptoms is to be relied on for the diagnosis, a single sign does not suffice.

Of especial importance is the determination of all conditions which make the diagnosis between pneumonia and *pleural effusion,* of a serous as well as of a purulent nature, certain. The more numerous the experiences of the individual physician are, the more he will be convinced that in this respect special attention must be devoted to the subject. Especially difficult may the conditions be in hospitals, because the patients are admitted in the various stages of the disease and are frequently seen in a condition in which it is not even possible to elicit an exact history. In private practice the diagnosis is easier if the patient has been watched and examined from the onset of the affection.

In all cases the diagnosis is naturally between *pleurisy* and a *pnemonia of the base,* especially in reference to an entire side. It is true that Traube once saw a pleurisy associated with a pneumonia affecting the same upper lobe, but such a curiosity need not be taken into consideration.

In the first place, even at the onset of pneumonia diagnostic difficulties may arise, especially if stitches in the side introduce the affection without chill; and neither crepitant râles occur favoring pneumonia nor friction favoring pleurisy. The latter may even occur at the onset of pneumonia. Only high fever, rapidly developing, is in favor of pneumonia; one single pneumonic sputum confirms the diagnosis. Especially important is the result of the examination of the apices of the lungs and the determination whether the patient has previously been healthy, especially whether he has ever suffered from cough. Pleurisy readily arises in connection with a feebly developed apical affection which up to that time had shown no symptoms. Under such circumstances the diagnosis between pneumonia and pleurisy may remain in suspense for one or even two days; after that time, however, either all the phenomena may entirely disappear; the dulness, the soft bronchial breathing, the increased pectoral fremitus, which is often absent in women and children, disappear as well as the fever, and convalescence follows; we have then had to do with a pneumonia which has lasted from two to three days. Pleurisy is excluded, as dulness due to that cause would not disappear so rapidly. Or the dulness remains and even increases. If slight fever is present, as is almost always the case; if bronchial breathing is constantly low and soft, as if it were only heard from a distance; if the dulness has not very rapidly taken in the entire base and its upper limit is lower than the upper limit of the lower lobe, or if it very gradually rises above that level, then we are dealing with a pleurisy.

But if the dulness has very rapidly taken in the entire base and has constantly remained limited to this area, an occasional loud bronchial respiration must suffice to make a diagnosis of pneumonia, especially in those cases in which hoarseness makes it impossible to test the pectoral fremitus and to determine bronchophony.

While these points may afford a slight help for the diagnosis during the first few days of the illness, it must be borne in mind that an effusion into the pleural sac may occur during the existence of a pneumonia. Usually the bronchial breathing, which at first was quite loud, gives place to soft continuous bronchial breathing. In such cases, fortunately, the failure to note this phenomenon is not of serious consequence; the pleural effusion usually disappears with the cessation of the pneumonia.

Only when a pneumonia occurs in the course of other acute diseases, especially in the course of enteric fever, the long continuance of dulness is to be carefully watched, and the diagnosis of "pleural effusion" confirmed by exploratory puncture, as the effusion might be purulent.

In the third place, a dangerous pleurisy may develop immediately after the cessation of the pneumonia; that is, just after the crisis. Such an event has an important influence on the subsequent course of the disease. The effusion is not infrequently purulent, and failure to make an accurate diagnosis may therefore be productive of fatal consequences. If the crisis occurs on the seventh or the ninth day, and is followed not only by a postcritical evening rise in the temperature, but by renewed fever lasting several days; or if, without the occurrence of crisis, the patient feels comparatively comfortable on or about the seventh or the ninth day, but later again develops fever with dyspnea and some cyanosis, while no signs of a secondary attack of pneumonia are discoverable in any other portion of the lungs, and the original dulness persists for several days longer, with coincident weakening of the respiratory murmur,—the diagnosis of a complicating pleurisy must be made, even if it should be impossible—on account of hoarseness, for example—to test the pectoral fremitus. A pleurisy of this kind is very often purulent; but the nature of the effusion must absolutely be determined by exploratory puncture, and the more dangerous, or at least the more disquieting, the symptoms after the seventh or ninth day of the disease, the more prompt must be the resort to this procedure. Naturally, it is to be wished that the puncture should always give a positive result, enabling one to determine whether the fluid be of a serous or purulent nature, but no one will presume to say that it has always been possible for him to do this. Even when an effusion is actually present the first puncture may be without result. Such failures are especially due to circumscribed agglutination of the pleura. Through this the exudate is prevented from gravitating to the lowest part of the thoracic cavity.

As regards the method of making an exploratory puncture, I have some suggestions which may perhaps not be without value to one or the other of my colleagues. In the first place, I do not use too short a needle. It is best to use a puncture needle made for the purpose, and longer than that which is usually found in the Pravaz syringe. The latter is too short to enter the thoracic cavity in corpulent patients. That it is well before using the needle to boil it and to disinfect the syringe with a 5% solution of carbolic acid before and after every use is obvious, but one should never puncture without cleaning the syringe in which carbolic acid has been present with a solution of boracic acid. Even a small quantity of carbolic acid which may have remained in the syringe may make the purest serous exudate cloudy, and so influence our judgment. The choice of the puncture site is, in the first place, determined by the dulness; in large exudates it is well to introduce the needle in the axillary line.

Now, however, after the most intricate conditions which are necessary for the diagnosis have been mentioned, it remains to be briefly stated that the displacement of neighboring organs—the liver, the spleen, the heart—materially facilitate the differentiation of pneumonia from pleurisy. One must, however, remember that such displacements are very frequently absent. One must not neglect the fact that a displacement of the heart, in spite of the presence of an exudate, need not be due to this cause but may even be due to a pneumonia of the left upper lobe, as the case previously quoted (page 471) proves.

[Pneumonia is sometimes mistaken for appendicitis. Pain in the abdomen, and even in the right lower quadrant, may occur in pneumonia, but, with high fever and vomiting, suggests the likelihood of appendicitis. The increased frequency of respiration and the expiratory grunt suggest a pneumonic origin for the abdominal symptoms. The latter symptom especially occurs in childhood, when, also, appendicitis is liable to occur.—Ed.]

NOTE ON BACTERIOLOGIC EXAMINATION OF THE BLOOD IN PNEUMONIA.

[Sereni in 23 cases found diplococci 3 times; White, 3 in 10 fatal cases out of a group of 19. Kohn, in 32 cases, secured the specific coccus in 9 only. Kohn thinks the absence of pneumococci from the blood a very bad sign.

White held that a general pneumococcus infection can be demonstrated occasionally in the late stages of acute lobar pneumonia.

Kühnau secured cultures on agar plates from 1 only of 9 patients. He inoculated animals with the blood, and twice in the 9 cases infection took place.

Baduel does not consider the presence of pneumococci in the blood as unfavorable. He recovered them 55 times out of 57 patients, in some instances as late as twenty-five days after crisis.

Krauss and Sello think the pneumococcus is only found in very bad cases.

Prochaska used 4 to 5 c.c., and Fraenkel 6 to 10 c.c., of blood on bouillon and recovered the diplococcus in every case.

Prochaska examined, in addition to 10 previously described, 40, all of whom had diplococci. Two had streptococci and diplococci: one died; the recovery of the other was much drawn out. He wonders if these streptococci are really streptococci, or only subdivisions of pneumococci, as they often form chains. Two had staphylococcus and diplococcus: both of them died; both had metastases. One had meningitis and ulcerative endocarditis as complications; one had a beginning lung abscess; one had serous pleurisy, another purulent exudate. In four the pneumococcus alone was found. He found them early in the disease; in one case as early as the second day. The number of pneumococci is not inverse to prognosis; some very light cases had very many, and vice versa. Why some should have toxemia, others not, he does not explain. Of the 40, eight died. That he found pneumococci in each case is due, he thinks, to the large amount of blood he used. He concludes that the blood of pneumonia patients always contains pneumococci; he has never found the Friedländer organism.

Orthenberger* found pneumococci in "smears" from 6 fatal cases. Perrine and Allessi† found them in 2 cadavers. Belfonti‡ found them in cultures from 6, and Banti§ in cultures from 18 out of 29 pneumonia patients. Klemperer‖ did not find any in 5 patients. Casati** injected rabbits with blood from pneumonia patients; all of them died of septicemia. Sittmann†† found them in 6 out of 16 patients and Brodie, Rogers, and Hamilton‡‡ in seven. Pane§§ says they are the exception and only occur in fatal cases; if accidentally patients should recover, pneumococci are weak and of diminished virulence. Berghini[*] found them in 8 cases, and believes them constant, regardless of gravity of case. Passler[†] found them in 6 fatal cases; in 38 cases who recovered he found them but once.

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* *Münch. med. Woch.*, 1888, 49 and 50.

† *Centr. f. klin. Med.*, 1890, 48.

‡ *Riforma medica*, 1890, 769.

§ *Lo sperimentale*, 1890.

‖ *Berl. klin. Woch.*, 1891.

** *Lo sperimentale*, VII.

†† *Deut. Arch. f. klin. Med.*, LIII.

‡‡ *Lancet* 1898.

§§ *Rif. med.*, 1899, III.

It is evident, then, that with good technique, using a large amount of blood, the specific organism of Fraenkel can be recovered in all cases of pneumonia, and that in obscure cases this may be a diagnostic method of positive value.

On Agglutination or Serum Reaction in Pneumonia.—Besançon et Griffon[‡] have found in animals suffering from experimental chronic pneumococcus infection, and in human beings ill with pneumococcic disease (pneumonia, pneumonial sore throat), a reaction comparable to the Widal reaction; that is, their blood is capable of agglutinating pneumococci. It is comparatively slight only, however. It acts only in undiluted or very slightly diluted state. To demonstrate this reaction, cultures of pneumococci are made in the serum. If the animal is healthy, the growing pneumococci form diplococci or short chains, clouding the medium diffusely; if the animal has pneumococcic infection and the agglutinating power is great, they form a thick tenacious deposit composed of chains of cocci without clouding of serum; if serum is weakly agglutinating, the cocci grow, clouding the serum slightly; to the naked eye there seems no difference from normal serum, but when examined microscopically we see, instead of short chains or diplococci, piled-up masses of cocci or chains of them. Thus we see that the differences between normal and many pneumococcic sera are not very great. Another factor which diminishes its clinical value is that the blood may agglutinate only pneumococci of the genus which have produced the infection, and not any pneumococci. We must, therefore, first cultivate the cocci from the individual and then try the agglutinating test. In most cases before this can be done the diagnosis has been made; in some sore throats, however, this test may be of great value, and examples of this are noted. The test is, however, of greater scientific than practical value. In cases of pneumococcic septicemia the reaction did not develop.

On Leucocytosis.—Williamson,[§] to determine if any connection existed between the leucocyte curve and the general state in pneumonia, infected a number of rabbits; from his results he sums up as follows:

1. The leucocyte curve begins with a sudden steep onset, and soon afterward sinks again; it falls to a depth considerably below that existing before the infection.

2. The height of the curve varies very much; occasionally it is entirely absent. No relation exists between leucocytes before infec-

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[*] *Clin. Med. Ital.*, 1899.

[†] *Münch. med. Woch*, 1901, 899.

[‡] "Etude de la réaction agglutinante du sérum dans les infections expérimentale et humaines à pneumocoques," *Ann. de l'Institut Pasteur*, 1900, xiv, 449.

[§] *Ziegler's Beiträge*, 1901, xxix, "Verhalten der Leucocytose bei der Pneumococcenerkrankung der Kaninchen u. Menschen."

tion and height of rise. Leucocytosis never lasted long, probably owing to great virulence of material of infection.

3. Hypoleucocytosis was always very considerable.

4. No connecting relation could be traced between the highest and lowest number and the pre-existing number of leucocytes.

5. No connection could be traced between the height of the initial hyperleucocytosis and the degree and time of the hypoleucocytosis and the clinical picture or the duration of the illness from time of onset to death.

6. If highly virulent pneumococci are injected in very small quantities under the skin, bacteria can be found in the blood within a very few hours. They may later disappear. They are destroyed by the antitoxin properties of the blood. This is proved by the fact that pneumococci cultured from the blood after the infection show feeble growth.

7. A distinct relation exists between the pneumococcus sepsis and hypoleucocytosis. Within a short time after the entrance of the pneumococci into the circulation the leucocyte curve begins to fall to far below normal. As this is also true in the croupous pneumonia of man, he considers this an explanation for the statement, clinically determined, that the prognosis of the disease becomes worse when leucocytes diminish in number. The absence of a hyperleucocytosis in many fatal cases of pneumonia he believes to be due to the presence of pneumococci in the blood.—Ed.]

NOTE ON THE URINE IN PNEUMONIA.

[Kleinmann * reports a case which during the first few days developed nephritis, and pneumococci were separated from the urine at height of the disease. He thinks, therefore, that nephritis is not an accidental complication, but due directly to the pneumococcus.

In pneumonia peptonuria was found by Gerhardt,† Meissner ‡ (after the crisis of each case), Brieger,§ Robitcheck,‖ Senator ** (a short time before and after crisis), Leick †† (each case), and Binet‡‡ (5 cases). Leube and Salkowski did not find it.

Ito§§ has found peptonuria in 7 out of 8 cases. It appeared several days before resolution began and disappeared several days after crisis. Brieger,‖‖ Naunyn,*** and Senator ††† have found the same. Jaksch ‡‡‡ found it only after crisis. Ito's paper is for the purpose of showing that a pure peptone may exist in human urine, which cannot be removed by saturation with ammonium sulphate. He does not mention the value of peptonuria.

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* Diss. Berlin, 1898.

† *Deut. Arch. f. klin. Med.*, 1869, v.

‡ *Prager. Quart. Jahrschr.*, 1879, CXLI

§ Dissert. Breslau, 1888.

‖ *Deut. f. klin. Med.*, 1894, XXIV.

** *Deut. med. Woch.*, 1895.

†† *Deut. med. Woch.*, 1896.

‡‡ *Rev. m. d. l. S. rom.*, 1890. 9.

§§ *Deut. Arch. f. klin. Med.*, 1901, LXXI, 29.

‖‖ Diss. Breslau, 1888.

*** *Arch. f. experim. Path. u. Pharm.*, XVIII, 67.

††† *Deut. med. Woch.*, 1895.

‡‡‡ *Zeit. f. klin. Med.*, VI.

Pick[*] calls attention to the fact that the urine of pneumonia twenty-four to forty-eight hours after the crisis changes its reaction, becoming neutral, amphoteric, or even alkaline; this takes place suddenly and is replaced after several days again by acid reaction. He observed 54 cases; 42 showed this phenomenon. Those which did not show it were either light cases, terminated by lysis, or women with previously alkaline urine. He believes this to be due to the absorption of the exudate, and chiefly because of the increase of excretion of sodium through the urine.

Dunin and Nowaczek[†] refer to Kossel's work, who demonstrated that the xanthin bases of urine arise from the broken-down nuclei of cells (nuclein) and the close affinity of uric acid to xanthin, hypoxanthin, and guanin; to Horbaczewski's work[‡]: he macerated spleen in distilled water at 50° C. (122° F.), producing slight putrefaction; he precipitated the albumin, added fresh arterial blood, and procured after several hours at 50° C. (122° F.) uric acid; leaving out blood and evaporating it, instead of uric acid he procured xanthin. The maceration fluid contained neither, but rather two bodies capable of forming either one or both: with oxidation, uric acid; without oxidation, xanthin; with insufficient oxidation, both. These papers prove that uric acid and xanthin are derived from the nuclei of cells. The feeding of nuclein increased the uric acid of animals; leucocytosis (physiologic or pathologic) always increased the uric acid. They also refer to Richter and Kühnau's work,[§] who fed nuclein (gr. v to x) to animals with resulting increased uric acid. Mayer[||] fed but 2 gr. and received negative results. Weintraub,[**] Umber,[††] and Mayer[‡‡] fed cellular organs (thymus, liver, etc.) and increased the amount of uric acid; in all 5 experiments no leucocytosis was produced; broken-down leucocytes did not produce uric acid in these cases, but this was formed directly from nuclein. Richter did not find leucocytosis invariably, wherever increase of uric acid was noted, but Kühnau did.

Dunin and Nowaczek observed 5 cases of croupous pneumonia regarding uric acid elimination. They noted an increase of elimination beginning with the day preceding the crisis, suddenly becoming very high on the day of the crisis (amounting to three times as much as ordinarily noted); it remains high for from two to four days after crisis, diminishing slowly, but not reaching the normal point until the seventh or eighth day after crisis. They base this upon a breaking-up of the exudate, many cells being excreted. Ranke[§§] and Gardes[||||]

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[*] *Deut. Arch. f. klin. Med.*, 1900, lxviii, 13, "Epicritische Aciditätsabnahme des Harnes bei croupöser Pneumonie."

[†] *Zeitschr. f. klin. Med.*, 1897, xxxii, 1, "Ueber Harnsaüreausscheidung bei croupöser Pneumonie."

[‡] "Siztungsbericht der Wiener Akademie der Wissenschaften," 1891.

[§] Resp. *Zeitschr. f. klin. Med.*, xxvii, xxviii. [||] *Deut. med. Woch.*, 1896.

[**] *Berl. klin. Woch.*, 1895. [††] *Zeitschr. f. klin. Med.*, 1896, xxix. [‡‡] *Loc. cit.*

[§§] "Beobacht. und Versuche über die Ausscheidung der Harnsaüre," 1898.

[||||] "Ueber Stichstoff und Harnsaüre ausscheidung bei verschiedenen Krankheiten," 1890.

noted the same thing. Otherwise the observers know of no occurrences. Bartels in 1866 casually mentions the same, as does Baftadowsky.

Petren* quotes Garrod,† who found uric acid in the blood of one pneumonia patient; Salomon,‡ who found it in 4 cases; von Jaksch,§ who found it in many cases and says it is due to a change in red blood-cells, which are not able to further oxidize uric acid after its formation from nuclein. Petren thinks this must be wrong, as it would have to be associated with increased uric acid excretion—a condition which does not exist. Klemperer‖ could not find it. Petren found large quantities in one case easily demonstrable by the murexid test. He says it is found almost always in blood of pneumonia patients and occurs frequently in normal man. He thinks that the cause of increase of uric acid in the blood of pneumonia and other diseases is due to diminished excretion by the kidneys and not to increased formation.

Kühnau** studied 6 cases of pneumonia, two of them traumatic. All had leucocytosis, but not proportional to their gravity. It remained for a few days after crisis, returning gradually to normal. Uric acid excretion during the period of the fever is above normal, but it continues to increase even after crisis, reaching its highest point several days after and at a time when leucocytosis is rapidly disappearing. Uric acid excretion then reaches normal quickly. He believes that the breaking-down of leucocytes is the cause of the increase in excretion of uric acid. Richter†† reports a similar observation among other conditions.

Douglass‡‡ observed 5 cases of pneumonia with regard to uric acid elimination and leucocytosis. All had increase in uric acid; only two had leucocytosis. Striking instances can be found where the two seem to go hand in hand, but the converse is also true. He does not believe that the normal seat of uric acid formation lies in the nuclei of the leucocytes, although he does not deny that cellular tissue (thymus, liver, etc.) increases it. He supports Horbaczewsky's theory, that uric acid is formed from broken-down proteids containing nuclein, especially leucocytes.—Ed.]

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* *Arch. f. experim. Path. u. Pharm.*, 1898, XLI, 265.

† *Medico-Chir. Trans. Lond.*, XXXVII, 181.

‡ *Zeitschr. f. physic. Chemie*, 1878, II, 65.

§ "Ueber die klin. Bedeutung von Harnsaüre und Xanthinbasen im Blute," 1891.

‖ *Deut. med. Woch.*, 1895, 655.

** *Deut. Zeitschr. f. klin. Med.*, 1895, XXVIII, "Verhältniss der Harnsaüre ausscheidung zu der Leucocytose."

†† *Deut. Zeitschr. f. klin. Med.*, 1895, XXVII, "Ueber Harnsaüre ausscheidung u. Leucocytose."

‡‡ *Edinb. Med. Jour.*, 1900, p. 32.

THE SPECIAL METHODS OF TREATING PNEUMONIA.

Venesection has been in practice in the treatment of pneumonia since the earliest times. According to Köhler, Hippocrates makes mention of venesection in the treatment of pneumonia of the lungs. Sydenham declared that repeated venesection, taking each time large amounts of blood, was a method which was absolutely indispensable, in the cure of the affection. Bouillaud carried this method to the extreme. He advises, in a pneumonia of medium extent, in the first or at the utmost in the second stage, in the case of an adult of usual strength and constitution, to take at least from 2 to 2.5 kg. of blood. To do this, general and local venesection had to be done continuously for three to four days after the admission of the patient. As a rule, he practised venesection three, four, or even five times, both general and local blood-letting (venesection of about 15 ounces, or 30 leeches, or wet-cups removing an equivalent quantity of blood), in the first twenty-four hours.

Louis was the first to oppose this bad practice upon the basis of his observations. He declared that venesection in the treatment of pneumonia was without value. It remained for Dietl to close the era of blood-letting. He demonstrated that venesection does not prevent the formation of the pneumonic exudate, that it is not possible to retard the pneumonic process in the stage of congestion and stasis or to repress it in the least degree; nor does this method of treatment shorten the stage of red hepatization. He believes, moreover, that as a result of venesection more massive hepatizations occur, and in those having died of pneumonia greater fibrinous plugs were found in the cavities of the heart and in the large blood-vessels. For this reason he even declares venesection to be harmful. In 17 fatal cases treated by venesection the pneumonia was complicated by meningitis three times, and five times with pericarditis; whereas in 22 fatal cases treated with tartar emetic only once was meningitis seen; and in 14 cases treated with dietetic measures no fatal cases occurred. The mortality as regards treatment was as follows:

With Venesection.	With Tartar Emetic.	With Dietetic Measures.
20%	22%	7.4%
17 out of 68	22 out of 84	14 out of 175.

The expression of Dietl: "According to our experiences, venesection is never indicated in pneumonia—that is to say, it is never necessary to bring about a cure," will nowadays be indorsed by the majority of physicians. Not by all, however. According to a vicious circle which not rarely occurs in science, the tendency to take up venesection is again making itself felt. It has even been advised—*horribile dictu*—at the end of the nineteenth century to employ venesection as a treatment for chlorosis! *

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* Even if venesection were harmless in chlorosis, it should not be employed, because we have many simpler methods at our command.

Huss discards venesection, just as did Louis and Dietl, in the stage of hepatization, and even declares that it is harmful to employ this method in that stage. He only advises the procedure to prevent the congestion from changing to exudation; that is to say, to prevent red hepatization. Whether he has ever accomplished anything with this method I regard as extremely unlikely.

According to past experiences, there appears to be only one indication for the employment of venesection—namely, the occurrence of edema of the lung in pneumonia as a complication in the earlier stages of the disease, provided that there be no decided weakness of the heart muscle, which would be a contraindication. Even if anything may be obtained in this manner, I must doubt it, according to my observations. The two cases of this sort in which I employed venesection in the first twenty-four hours of the disease, on account of edema of the lung, terminated fatally within the first thirty-six hours. The autopsy confirmed the diagnosis. Talamon discards venesection entirely. He says: "We do not bleed in acute rheumatic fever, in typhoid, in erysipelas; why, then, retain this barbaric procedure in pneumonia?"

Not less historic than the use of venesection is that of its competitor, tartar emetic. Advised by Rasori, and used in enormous quantities, this remedy, through Laennec's authority, remained in use a long time, in spite of the proof that the statistical reports given by him were not reliable (compare Grisolle, p. 622). A complete historical review in reference to the different modifications and modes of employment, and of the combined employment of tartar emetic and venesection, is given by Köhler; nowadays the use of tartar emetic has been almost entirely abandoned. Talamon quite properly drives the last nail into its coffin with the words: "We diminish the chances of cure with its employment in that we complicate the disease with the effects of a fresh intoxication."

Aran introduced veratrin into practice, and has found warm adherents in Biermer and Köhler (compare Köhler, I, p. 798). To thoroughly frighten off any one from the use of this drug I advise them to read the twenty-five histories of the cases given by Alt. Von Jürgensen is right when he says that only ignorance of the normal course of pneumonia has led to the employment of veratrin.

Pilocarpin has also been employed. Sziklai has given adults from 3 to 4, children over four years of age from 1 to 2 cg. per day, and believes that he has achieved good results. Liszt believes the same. Richter, on the other hand, has not noticed any influence upon the pulmonary inflammation.

An exceedingly rich literature is at hand in reference to the employment of digitalis in pulmonary inflammation. The first employment of this drug also dates from Rasori, and the school of contrastimulists. Later, digitalis was more frequently employed on account of the investigations of Traube. However, it cannot be said that this treatment was especially advised by him; at least what he says in reference to this drug does not sound very encouraging. He employed the drug in very large doses (120 grains in forty-eight to sixty hours). According to his observation, this produces a very great diminution in the pulse frequency, but simultaneously with the decrease in the tension of the arteries no increase in diuresis is noted. Very frequently, however, decided collapse was seen. There is here, as he supposes, besides a stimulation of the regulating apparatus of the nervous system, a beginning paralysis of the musculomotor nervous system.

Lately digitalis has again found a very eager champion in Petrescu. Doses which sound enormous are employed by him. He gives from 12 gm. in infusion up to 200 in twenty-four hours. According to his opinion, provided these large doses of digitalis are given early, they will abort every typical pneumonia (in atypical pneumonias the course is not so favorable) and bring down the mortality of pneumonia to zero. He has up to this time, so he reports in his first statistical publication, had but a mortality of 1.21%, as too many of his patients were treated too late.

In his later communications[191] he has not had less, but more, fatal cases. Up to the 1st of July, 1887, he had 577 cases, with a mortality of 1.21%; up to January, 1889, he had 816 cases, with a mortality of 2.06%; up to the year 1891, he had 825 cases, with a mortality of 2.06%. It must, however, be remembered that these cases were treated in a central military hospital, in which he made his observations.

To attain such results it is by no means necessary to employ digitalis as a treatment. In my mortality statistics previously quoted I had in 379 cases of pneumonia in ages ranging from five to twenty, 11 deaths, which gives a mortality of 2.64%. If I reflect that my patients were taken from the civil population, which were only compelled by dire necessity to take up treatment in a hospital, and that quite a large percentage at the end of their disease were sent to the hospital moribund, I should be very nearly inclined to declare the cases treated by me without digitalis as the more favorable ones.

More conspicuous even are the figures given by Risell. How can one make a great fuss about the treatment by digitalis if this author, without digitalis, in 127 pneumonias in persons in the second and third decennium of life, has shown but 2 fatal cases, therefore a mortality of 1.8%. Barth declares the employment of digitalis in large doses is useful in cases in which baths are contraindicated or cannot be employed. He protests energetically against those theoreticians who, under the supposition that pneumonia is a cyclical disease, desire to bring the expectant method into treatment again. Without doubt the simple pneumonias, such as occur in children and robust young people, in a majority of the cases do not require active treatment, and just so all the methods of treatment in the world would fail to prevent the fatal issue of pneumonia when it occurs in diabetics, uremics, and cachectic individuals, and in all cases in which pneumonia is the final event in life. But between these two extremes there are still numerous cases in which the prognosis remains undecided, and in which the battle for a long time is in doubt, and in which proper methods of treatment will produce a favorable outcome of the affection.

In all cases of pneumonia in which the extension of the hepatization, the intensity of the fever and dyspnea, the frequency and softness of the pulse, make us fear cardiac weakness, one dare not dally in the use of proper therapeutic measures. If the patient be robust, has not reached or overstepped the fortieth year, if there be no serious organic defect, digitalis should be given the preference over all the usual methods of treatment: 1.5 to 3.0 gm. in infusion in 100 gm.,—that is, 25 gm. rum and just as much syrup of orange peel, —and allow the patient to take a tablespoonful every two hours. We must carefully watch the action of the remedy, but should not allow ourselves to be frightened off from employing it by slight vomiting or vertigo. In suitable cases alcohol and hypodermic injections of caffein may also be employed. The use of digitalis need not be stopped unless the pulse becomes very slow or irregular; else, according to indications, it should be used for from four to five days, until the fever has disappeared. Fickl gives daily doses of 3 gm. in infusion as long as fever is present, or slight intoxication phenomena are noted. With this he employs stimulants plentifully. Of 108 soldiers sick of pneumonia, 93 recovered completely and were able to continue their service, 13 were given leave of absence to convalesce completely, one was transferred after convalescence, one died. Three fatal cases, however, were excluded from this count on account of complications.

Von Jürgensen is opposed to the use of digitalis in so far as a specific action is supposed to be due to it. He believes, however, that under certain circumstances it is of value, which he describes minutely: "In a person previously suffering from cardiac weakness, or who in the course of the disease develops cardiac insufficiency, if the pulse without obvious reason becomes greatly accelerated, and at the same time irregular, digitalis is indicated. The indication or contraindication depends upon the pulse remaining full or becoming empty; if the volume is good, then one may expect something from the remedy, especially toward the end of the disease, in which one of the favorable signs is not rarely a slight irregularity of the pulse."

Reiner reports from Professor Drasche's clinic 24 cases treated by digitalis, of which but one, a man aged sixty-nine, died. From his experiences it may be noted that the daily dose may be raised without danger over the usual doses of digitalis, and that the fear of large doses in general is not justified; only these doses must not be continued longer than two days at the utmost. His observations show neither a decided action of the digitalis in pneumonia nor a shortening of its course. On the contrary, he believes that resolution was delayed by the treatment. Neither was it possible to bring down the temperature by means of digitalis. The pulse frequency was not influenced during the hyperpyretic stage; only in the period of apyrexia the pulse fell to about 50, and then in several of his cases collapse occurred. In short, nothing can be adduced to justify the employment of large doses of digitalis in pneumonia.

In a similar manner Gerhardt declares that digitalis in his hands did not come up to his expectations, and Talamon says: "If digitalis is contraindicated in large doses in severe cases of pneumonia, as Petrescu and Fickl admit, why should they be employed in such cases as recover of themselves?"

I myself, under the influence of the teaching of Traube, some years ago employed digitalis in infusion of 2 gm. in 180; but once after two bottles, in a woman of thirty years of age, have seen a fatal collapse, which I could only explain by the action of digitalis. Whether the circumstances in private practice—that is, the impossibility of constantly observing the course of the disease during the use of this remedy—have had an influence, and whether such an event could occur in a hospital, I must leave undecided. Since that time I have no longer employed digitalis, and would not even now be induced to use digitalis in the hospital, as, especially in the last years, I have been enabled to obtain very satisfactory results by less severe methods. The only indication for the employment of digitalis I maintain to be an irregularity and rapidity of the pulse due to a previous myocarditis,—which usually depends upon an atheromatous degeneration of the vessels,—especially occurring in elderly individuals. By the employment of an infusion of 2 in 180, of which a tablespoonful is given every two hours, I have occasionally, after the use of one to two bottles, seen favorable results.

A great part is played—perhaps we shall soon be able to say has been played—by *alcohol* in the treatment of pneumonia after Todd had advised its employment in febrile diseases generally. After him, as a decided opponent of the active antiphlogistic treatment, and as an upholder of the dietetic and stimulating treatment, Hughes Bennet arose. By his treatment the mortality was reduced so much that but one fatal case occurred in 35 pneumonias. In the "British Medical Journal" of April 14, 1886, it was proved to him that for some reason he had omitted the fatal cases (Köhler, I, p. 773). Nevertheless the adherents—I may say of forced feeding with alcohol—increased. Sée declares that clinical experience has justified and experimental physiology confirmed the bold procedure of using large doses of alcohol in the treatment of fever. This is even to-day the most rational procedure, because (1) alcohol serves as a directly absorbable nourishment without previous change in the veins; (2) alcohol maintains the strength by diminishing great loss of tissue; (3) it maintains strength also by its direct action upon the neuromotor system; (4) it acts as an antithermic agent, and perhaps also as an antipyretic agent.

Von Jürgensen is also an unqualified adherent of the alcohol treatment. He gives alcohol in every pneumonia, "not according to rote, but according to distinct indications which must be decided in each individual case." Every adult patient suffering from pneumonia has administered to him daily from the start one liter, a child, according to age, $\frac{1}{4}$ to $\frac{1}{2}$ a liter, of mild good red wine. In the treatment of pneumonia with cold baths, before and after the bath 30 to 50 c.c. of red wine are administered; in very cold baths not too small a quantity of some heavy wine,[118] such as wines of the South, Hungarian wine, Burgundy.

According to his experience, the patient as long as the fever lasts will absorb quantities of alcohol which in a normal person would certainly produce drunkenness, and perhaps even the danger of severe intoxication.

I myself am decidedly opposed to the use of alcohol in the manner described. Under the stress of public medical opinion I also employed the treatment for a few years. But as during this time the results were comparatively unfavorable, and at least no positive advantage appeared to be gained; as, moreover, no distinct diminution of tissue-waste was demonstrated by my weighing experiments, and I never observed any antithermic or antipyretic effects in pneumonia, I gave up the routine use of alcohol. I cannot help believing—on subjective grounds, it is true—that the ingestion of half a bottle of Hungarian wine every day for several days in succession is capable of converting excitation into prostration; in some cases I had a distinct impression that the stuporous condition of the patients was due to alcohol inoxication.

I do not mean to say, however, that alcohol should be given up altogether. Occasionally I also have found it necessary to resort to alcoholic stimulants; less frequently in private practice than in my hospital service. Patients in good circumstances, when they are attacked with pneumonia, have a much larger reserve of albumin, and, as a rule, do not need alcohol at all. But in hospital work the patients when admitted are often much reduced in strength, and may with perfect propriety be given a moderate amount of alcohol. The only other indication for the administration of alcohol is found in conditions of collapse.

Cold baths have also been used in the treatment of pneumonia. Fortunately this method of treatment has so far become even less general than the use of alcohol. Fismer has reported the results of this method of treatment, which was first proposed by Liebermeister, as carried out in the hospital at Baden. The temperature of the bath was 16° R. (68° F.), the duration ten minutes. For elderly or very debilitated individuals the graduated cold bath advised by Ziemssen, or baths at a temperature of from 20° to 24° R. (77° to 86° F.), were used, the patient remaining longer in the water. Of 230 patients treated in this manner, 38, or 16.5%, died; whereas before the mortality had been 25.2%.

Talamon severely condemns the use of cold baths in the treatment of pneumonia. He declares that they have no effect whatever on the pneumonic process. Even the supporters of the method admit that cold baths are not to be used when the process is extensive, when there is decided adynamia, when the respirations are markedly increased (Fismer), in the presence of arteriosclerosis, and in cardiac insufficiency (Barth). It is better to abandon altogether a method of treatment which is useless in cases in which it might be employed, and counterindicated in cases in which it might prove useful.

I myself have never resorted to cold baths in the treatment of pneumonia, although I have seen them used when in consultation with some colleague; but I have never been able to convince myself of their value. In some cases the irritative cough was increased by the cold baths. One objection to the use of cold baths, that has been raised on pathologic grounds, appears to me worthy of especial notice. Marchand voices the opinion that the tendency to the production of chronic induration of the lungs is increased by unfavorable temperature conditions. Although it has recently been claimed that the cooling of the skin in febrile diseases is quite harmless, and this view has revolutionized the treatment of febrile diseases, he nevertheless believes that sudden and continued withdrawal of heat is not a matter of indifference. If a man suffering from a recent attack of pneumonia spends the night in the open,—and persons have been found dead under such circumstances,—he subjects himself to a continuous reduction of temperature; the very frequent occurrence of induration in such cases may perhaps be attributable in a large measure to this abstraction of heat. In Marchand's case of induration of the lungs the patient, in addition to having been thoroughly drenched, was treated from the beginning with frequent cold baths. The lung in this case was found to be abnormal, "owing to already existing pleuritic adhesions at the site of an old induration in the apex." In the subsequent course of the disease a pericardial effusion was superadded. Marchand leaves it undecided whether, in view of the patient's previously acquired predisposition, the therapeutic measure so warmly recommended by v. Jürgensen was not in a large measure responsible for the unfavorable outcome. Especially when there is high fever, a marked reduction in the temperature of the skin in pneumonia, which is, as a rule, characterized by great turgescence of the skin, necessarily produces congestion of the internal organs. The antipyretic action is of little moment, as it practically affects only the integument. This view is supported by the experiments of Lassar, who, as is well known, demonstrated that intense cooling of the skin is followed by the production of interstitial inflammatory processes in internal organs.

Only when the temperature is excessively high, the extremities are burning hot, and the consciousness is clouded have I used tepid baths (26° C.—78.8° F.). In general the indications for the procedure are very few.

It follows, from what has been said in regard to former methods of treatment and those in use at the present time, that there is little prospect of ever discovering a uniformly successful treatment for pneumonia. Nevertheless we are not reduced to the expectant plan pursued by Skoda and Griesinger. Although it was shown by my statistics that, with a strictly expectant method of treatment, only 8 out of 81 patients, or 9.8%, died in the year 1880,—that is to say, until the last two years the most favorable mortality conditions prevailed during that year,—I cannot bring myself to adopt the expectant plan of treatment. For during the years immediately following 1880, I became convinced that the severity of pneumonia and the mortality of the disease are subject to marked variations in different years.

According to my experience, *quinin*, when properly used,—that is, in accordance with all the symptomatic indications, which in pneumonia are quite numerous,—is the most suitable remedy. The drug was long ago employed by France, Cordon, and Corrigan,* and has recently been recommended by Huss and v. Jürgensen. Huss uses it more as a tonic, and gives it in small doses (10 to 15 cg.—grains 1½ to 2¼—four to six times daily). Jürgensen, on the other hand, recommends quinin as an antipyretic; for adults in doses of 2 grams (30 grains) given in the evening between 6 and 8 o'clock, and for children 0.1 to 0.2 gram (1½ to 3 grains) for each year of life, but not more than one gram (15 grains) up to the tenth year. His experience shows that the full effect is obtained only when forty-eight hours are allowed to lapse between the doses, which must not be divided. The reduction of the body-temperature occurs slowly and amounts to, at most, 1.5° to 2.5° C. (2.7° to 4.5° F.); the temperature, however, remains slightly lower for the following twenty-four hours. To obviate the bitter taste, the remedy may be given by the rectum according to the following formula:

℞. Quinin bisulphate		2.0	
Tincture of opium		10 drops	
Decoction of althæa	q. s. ad	100.0.	M.

Sig.—To be given as an enema.

Sée gives a detailed description of the mode of action of quinin in pneumonia (page 255). The tolerance of the organism for quinin sulphate is limited to 2.5 grams (37.5 grains); if more is taken, fatal collapse may result. But unless 1.5 grams (22.5 grains) at least are given, the antipyretic effect is *nil*. "It is therefore advisable to follow this formula, and to give the entire amount in two doses, without dividing them." The doses given should bring about a reduction of the temperature in from five to seven hours; if it fails to take place, the case is one of the greatest gravity, and cannot be brought to a favorable termination by any method of treatment. By means of these undivided doses a reduction of 1.5° C. (2.7° F.) can be obtained in pneumonia, but after eighteen to twenty hours the temperature tends to regain its original level.

On the strength of these recommendations I experimented with doses of 1 gram (15 grains), but never observed the slightest influence on the fever. I therefore gave up this method of administration and, like Huss, confined myself to small doses of quinin; but I combined the quinin with iron, the use of which in pneumonia has been warmly advocated by Niemeyer. This drug was frequently ordered with very good results by my former chief at the Magdeburg-Altstadt Hospital, Medicinalrath Doctor Schneider, as I can personally testify. Believing that it should be our aim to render the taking of medicine as pleasant as possible for the patient, I have selected from among all the preparations of quinin the *neutral tannate*,* because it is almost entirely tasteless. As this preparation, according to Roznyay, is weaker than by two-thirds than the muriate, I give to adults 1 gram (15 grains) three times a day, combining it with 1 gram (15 grains) of ferrum oxydatum saccharatum. With this treatment it appears to me that the course of even moderately severe cases is, on the whole, satisfactory. That is not saying very much, it is true, and it is not this use of quinin that induces me to attribute therapeutic importance to the drug. In addition to these powders of quinin and iron, I have employed quinin in another way in severe cases of pneumonia during the past two years. I refer to cases in which insomnia and restlessness developed during the course of the disease, without pain; the pulse was rapid and small; and the mind clouded. The temperature in these cases was no criterion of the severity.

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* See Köhler, vol. I, page 800.

To elucidate the mode of action of quinin I shall quote the opinion of Finkler, which is, briefly, that it is incorrect to speak of quinin only as an antipyretic agent. As the drug influences the fever only in certain diseases, it follows that it owes this specific selective action solely to its effect on the various specific causes of fever. As early as 1870 Binz demonstrated (according to Finkler) that the antipyretic action of quinin cannot be explained by any direct influence on the heat-regulating centers. This is further shown by the fact that quinin acts in intermittent fever even when it is given during the afebrile interval, that it is an effective prophylactic (Gräser), and that it exerts some influence on the pus-corpuscles, limiting their emigration from the blood-vessels and the formation of pus, as Binz has demonstrated. "If quinin has any influence on those bacteria and their products which give rise to suppuration and to the multiplication and emigration of white blood-cells, it is probable that it also exerts some inhibitory influence on the bacteria of pneumonia which possess similar properties."

The value of quinin in the treatment of pneumonia, according to my observation, is due less to its antipyretic action than to its specific action on the causes of the disease or their products. When quinin is used after the method to be described in the section devoted to the treatment of pneumonia, I have in a few cases seen a reduction of the temperature, but much more frequently its effect on the general course of the disease is distinctly favorable.

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* Supplied by Roznyay in Arad.

Serum.—It remains for later investigators to find out if it will be possible, with the help of the antitoxins of the pneumococcus, to cure pneumonia and so make unnecessary all methods of treatment in use at the present time. Attempts of this kind have already been made; only the most important may be here mentioned.

Netter succeeded in immunizing mice and rabbits against the pneumococcus. He employed for this purpose the dried spleen of animals which had been infected with the pneumococcus; old pleural exudate containing pneumococci, and the sputum of convalescents after the crisis when it was no longer virulent.

From his careful investigations in regard to the causes of immunity Emmerich at the same time with Fowitzky concluded that pneumonia in animals (rabbits) can be cured by injecting the blood and tissue juice of rabbits that had been artificially immunized, and that an outbreak of the disease could be prevented if the animals, a short time after their infection with a pure culture of the special pathogenic bacteria, had injected into them the serum of artificially immunized animals. The tissue juice and the blood of artificially immunized rabbits show a marked difference in their action according to the method that has been used in preparing them. If the rabbits are immunized by means of subcutaneous injections of weakened cultures, an incomplete immunity is attained, and the serum prepared from these animals does not possess the highest curative powers obtainable. On the other hand, by the use of intravenous injections of diluted cultures having their full virulence complete immunity is produced and a serum of marked curative power is obtained.

F. and G. Klemperer injected 20 c.c. of a purulent pleural exudate that had been freed from other germs by repeated cultivation into two rabbits; both were inoculated after fourteen days and withstood infection, while the control animal succumbed. They were also able to immunize with rusty sputum obtained before the crisis, provided its poisonous qualities were removed by heating. The conclusions of their findings show that any culture-medium in which the pneumococcus has been grown will immunize an animal against pneumococcus septicemia, even after the cocci themselves have been eliminated. The immunizing action is increased and quickened if a culture-medium containing the cocci, or one that has been freed from cocci but still retains its virulence, is subjected for some time to a high temperature—60° to 65° C. (140° to 150° F.) for two hours, or 40° C. (104° F.) for three or four days.

The curative serum, however, does not kill the cocci; it only destroys the virulence of the products formed by the pneumococci.

In connection with the previously mentioned immunizing experiments on animals, F. and G. Klemperer report that they injected themselves with 0.5 to 3 c.c. of the serum taken from animals immunized against pneumococcus infection, and proved that the material is entirely harmless in normal persons. Following this, they injected 4 to 6 c.c. of such serum into 6 pneumonia patients. In all the cases, in from six to twelve hours after the injection, a decided fall in the temperature occurred with a slowing of the pulse and respiration; once the temperature reached 37° C. (98.6° F.); twice it remained constantly normal; in the first cases the temperature rose, on the average, after six hours.

Foà and Carbone used in a case of pneumonia in a human subject injections of blood-serum taken from a rabbit immunized by injections of sterile cultures. The first injection of 5 c.c. of blood-serum resulted in a decided drop in the temperature, simultaneously with a reduction of the pulse frequency and the respiration. On the following day, that is, upon the fourth day of the disease, after a similar injection of the same quantity of blood-serum, the crisis occurred. Without attempting to draw definite conclusions, they nevertheless call attention to the importance of recognizing the harmlessness of taking blood from an immune rabbit and injecting it into a human being, and to the possible advantages to be derived from perfecting this method of treatment.

In conclusion, for the sake of completeness it must be mentioned that the attempt has been made to utilize the leucocytosis that takes place in pneumonia as a therapeutic measure. In regard to the observation that those cases of pneumonia which occur without marked leucocytosis are the most unfavorable prognostically, v. Jaksch comes to the conclusion that it would be of advantage, in cases in which leucocytosis is absent, to use remedies subcutaneously or internally which have the power of increasing the number of white corpuscles circulating in the blood. He was able in one case to increase the number of leucocytes 62% by the hypodermic injection of pilocarpin, but no other particulars are given in reference to the case. Lähr, in a case of pneumonia with a low leucocytosis, was unable appreciably to increase the number of leucocytes by means of the cold pack.

PROPHYLAXIS AND TREATMENT OF PNEUMONIA.

If it were possible to prevent the entrance of pneumococci into the alveoli of the lungs the occurrence of pneumonia would be impossible. Ways and means to prevent this are, however, unknown. The advice to carefully destroy the sputum of a pneumonia patient is certainly correct, but we must not expect particular results from this measure if we reflect that pneumococci are extremely frequent in the oral cavity; they may even be found in the bronchi and in the alveoli of healthy lung.

Up to the present we are unable to prevent the entrance of the pneumococci; perhaps it may be possible by the use of serum of animals that have been immunized against the poison of the pneumococcus to at least produce a reduction of the deleterious effects of these bacteria upon the human organism. The fact previously demonstrated (page 505) of the difference in virulence of pneumonia in different years, independent of any special methods of treatment or other predisposing circumstances, proves that the action of the bacterial products must vary in different years and warrants the hope that it may be possible to bring about a condition of affairs which will render the harmful action of these germs less severe.

Measures to prevent the direct transmission of the disease from pneumonia patients to healthy persons, or to patients suffering from other affections, are useless, as no one has noted the direct transmission of this disease. In pneumonia more than in any other disease observations in the hospital are sufficient to exclude such transmission. Pneumonia patients are always placed among other patients, but there has never occurred to me or to other observers the slightest suspicion that any contagion had occurred.

As we are unable successfully to combat the diplococcus, the primary cause of the affection, we must direct our efforts against the auxiliary causes, or at least render them less active. But even here there are very narrow limits. Since a child in the course of its first two years of life may have three attacks of pneumonia and adults may be affected 5 times in the course of their lives, we must assume a predisposition on the part of the lungs of the affected individuals. Of what nature this predisposition is, in what it consists, we have up to the present time not the slightest idea. For this reason it is impossible to take measures to guard against it.

It only remains to take prophylactic measures against those causes whose action is a temporary one, of short duration, and whose influences may be referred to external causes. The effects of wet, cold, or the marked chilling of the surface of the body in one who has been subjected to severe bodily exertion, or in one who has weakened his constitution through excesses of any nature whatsoever, can be effectively combated, and in this manner we can remove a direct cause of the disease. It is true that the physician rarely sees the patient at such a time, but the knowledge of such conditions may be utilized much more than has been done up to this time. How frequently does one meet with individuals, even educated persons, who have not the slightest idea that it is extremely dangerous to life to expose themselves to a draft, after having exerted themselves by a tedious walk or a mountain climb, having overheated themselves and thoroughly wet their clothing with their own perspiration; that, moreover, it is absolutely dangerous to expose one's self while wet with perspiration to a draft, and, that if it is at all possible, the underclothing should be changed at once so as to avoid the abstraction of heat from the body incident to the clothing drying by evaporation. We are not at all astonished if a workman does not know that, after having been wet through and through by the rain while at work, it is well for him to change his clothing, or, if that is impossible, to remain in bed for a short time. In most cases he has plenty of time to do so, but prefers to spend it in the tavern in his wet clothes.

If under such circumstances the sensation of a general feeling of malaise and chilliness develops, then it is certainly advisable to go to bed at once, and by means of a cup of elder tea to induce active perspiration. Such advice may appear as of slight value; nevertheless if the advice be carried out, it will often prove of benefit.

Let us, however, take up the positive question of the treatment of the disease itself.

If croupous pneumonia begins gradually, with general malaise, headache, and chilliness, the physician is rarely in the position to give adequate advice. He has not even anything of importance to say, especially if he does not attach any importance to the above-mentioned causes which have preceded these events in many instances.

The *convulsions* which usually usher in the disease in children do not require special treatment, as they commonly pass off without leaving any bad effect upon the organism. Cold applications or, more properly, linen cloths rung out in ice-cold water may be placed upon the head; their effect is more to quiet the members of the family than to be especially useful to the patient.

Also for the *vomiting,* which in some few cases ushers in the disease, no special treatment is required. Should the vomiting be continuous, lasting several hours, something that I have never observed in pneumonia, it would be necessary to have recourse to a hypodermic injection of morphin.

The patient takes to his bed of his own accord upon the appearance of the *initial chill,* which is of such frequent occurrence at the onset of the affection. If it is at all possible, the bed should be previously warmed, especially in winter. This may be accomplished by various methods—hot-water bags, hot bottles, hot bricks, etc.; such a warmed bed is very grateful to the patient. Who has not felt the sensation of cold, even amounting to a positive chill, even when fever is present, on entering a cold bed? Furthermore, hot tea should be given in all such cases; this serves to diminish the sensation of cold.

The treatment must be more active for the *pains in the side,* which may appear simultaneously with or after the chill, especially if these pains be severe or interfere in the slightest degree with the respirations. I have never advised the use of leeches, which even to-day is a favorite method of treatment; neither have I advised wet cups. The only proper method of removing these pains in the side is by giving a sufficient dose of morphin hypodermically. In adults a dose of 1 cg. ($\frac{1}{6}$ grain), in young individuals between the ages of ten and sixteen 5 mg. ($\frac{1}{12}$ grain), and in younger children 2 or 3 mg. ($\frac{1}{30}$ to $\frac{1}{20}$ grain) will usually prove sufficient. Only very rarely must the injection be repeated in the course of ten or twelve hours on account of the pain. The pains after the first injection, as a rule, do not recur or are so mild that an ice-bag applied to the affected side is sufficient to relieve them. I can also recommend the hypodermic injection of morphin in private practice. Apart from the fact that the procuring of leeches or of wet cups requires some time, during which period the patient must continue to suffer, the result is very uncertain. Every physician, however, carries with him a hypodermic syringe and a quantity of morphin which he may employ at once without the loss of time. He should not, however, carry the morphin in the form of a solution, which is unsuitable when the drug is only used occasionally, but in the form of a powder in paraffin paper, each powder containing exactly 2 cg. ($\frac{1}{3}$ grain) morphin by weight (without sugar). He need only then at the bedside ask for a spoon or a small glass containing boiled water, or take a tablespoonful of cold water and hold it over a gas flame, pour the water into another spoon containing the powder, which easily dissolves in the liquid, draw it up into his syringe, and inject it under the skin of the patient. In this manner the hypodermic syringe contains a definite quantity of morphin, which is not so likely to be the case if a solution is employed, as frequently the caliber of the syringe varies and an unknown quantity of morphin is used. If the 2 cg. ($\frac{1}{3}$ grain) of morphin are taken up in solution into the syringe, each division of the syringe will contain 2 mg. ($\frac{1}{30}$ grain) of morphin. With the aid of the screw on the piston-rod of the syringe, the number of divisions to be given can be accurately measured off. My advice is to do this in every case; no matter how absent-minded the physician may be he will thus avoid giving too much morphin.

While the ice-bag which has been applied to the affected side on account of the slight pain is allowed to remain in place during the subsequent course of the disease, and a second one may be applied to the head on account of the headache, or cloths rung out in cold water may be substituted, there is now no distinct indication for the administration of drugs unless the patient has a previous myocarditis with frequent irregular pulse or a heart lesion with frequent heart's action. In such cases I believe the use of digitalis is indicated: 0.25 gram (4 grains) pulv. fol. digit. with 0.5 gram ($7\frac{1}{2}$ grains) saccharum as a powder, of which one should be taken 4 times daily; that is, inside of twenty-four hours 1 gram (15 grains) of pulv. fol. digit., or in infus., 1.5 grams ($22\frac{1}{2}$ grains) in 180 c.c. of water with the addition of 20 c.c. of syr. simpl., of which day and night one tablespoonful should be taken every two hours; that, in my opinion, is a sufficient dose to produce a slowing of the pulse in the course of two to three days.

This indication, on the whole, however, is quite rare. In patients in whom pulse and respiration are not too frequent, in whom the mind is clear, the cough not too severe, and no decided delirium occurs,—in short, in cases which every physician sees who frequently has the opportunity to observe cases of pneumonia,—there is no indication for treatment so long as no severe symptoms or complications make their appearance. These are the cases in which pure homeopaths,—I know such a one who calls himself a homeopath and gives his patients large bottles of a powerful morphin solution, which he allows them to inject into themselves,—hydropaths, and natural healers, celebrate their triumphs. If I, nevertheless, in the absence of distinct indications, advise the use of comparatively small doses of quinin and iron, just as other observers do, I am confident that this empiric mode of treatment is of value in inflammatory affections in which anemia is apt to occur. An exact definition, such as influencing the process of leucocytosis, preventing the migration of white blood-corpuscles, is nowadays no more than a paraphrase; it rests upon a too hypothetic basis.

I prescribe, therefore, provided there are no other indications:

℞. Chinini tannici neutralis..................
Ferri oxydati saccharati.................... āā 1.0 (15 grains).
Three times daily, one powder.

Younger individuals receive one-half of this dose; children, one-fifth.

I must, however, especially remark in this connection that chininum tannicum neutrale is not identical with the chininum tannicum officiale of the pharmacopœia. The latter has a bitter taste; the former is almost tasteless. It is obtained, as has already been mentioned, from the apothecary Roznyay in Arad (Hungary). One gram contains about as much quinin as is contained in 0.33 gm. chininum hydrochloricum. Hence, if the patient receives three doses of 1 gram each, he gets as much quinin during the day as is contained in 1 gram of the muriate. Regarding the ferrum oxydatum, I especially emphasize that it is a 10% preparation; else a preparation might be employed that contains a smaller percentage of iron. In regard to the administration of the powders, it is to be observed that they do not dissolve readily in water. If the patient cannot take them in wafers, or dry, with a mouthful of water, they may be advantageously added to a spoonful of apple-butter, thick soup, bread-crumb paste, or Hungarian or port wine.

In addition to this treatment, consisting of an ice-bag and the powders of neutral tannate of quinin with ferrum oxydatum saccharatum, some morphin will occasionally be required on account of the cough, especially if it be marked at night. Adults in such instances receive 15 mg. ($\frac{1}{4}$ grain) of morphin internally.

I distinctly advise against the use of stimulants. In private practice, especially among the well-to-do classes, if the patient was previously strong and healthy, stimulants may often be dispensed with during the entire course of the disease. The same is true of robust adults of the working classes. Only in patients admitted to the hospital in a debilitated condition, from previous causes of any kind, are stimulants useful. Under these circumstances a mixture containing spirit. rectificatus (90%) 40.0 (say, f℥iss), extr. aurant. 0.5 (♏viiss), syr. simp. 30.0 (f℥j), aq. dest. ad 200 (say, f℥vj) (for women and children diluted with a similar or double quantity of water), a tablespoonful twice daily, is sufficient. Instead, under favorable circumstances, Hungarian or port wine, and, according to the wishes and inclinations of the patient, champagne and beer may be employed. I treated an elderly colleague who was accustomed to the free use of beer by allowing him two mugs of beer, exactly 1 liter, each day during the entire course of his pneumonia. The beer was taken fresh from the keg. He recovered completely from his severe pneumonia. He succumbed eight years later to paralysis of the heart after several stenocardiac attacks.

Stimulants are temporarily necessary in combating attacks of collapse. In this condition champagne is most valuable. With this, the employment of solutions of camphor hypodermically is strongly advised. Of a mixture containing 6 gm. (ʒiss) camphoræ purissimæ in 24 gm. (fʒvj) oleum olivarum purissimum two to four syringefuls (0.4 to 0.8—⅗ to 1⅖ grains—camphor) may be injected at one dose. If one is careful to keep the syringe clean by cleansing it after each use with a 5% solution of carbolic acid, one need not fear the occurrence of abscesses at the point of injection. In thousands of injections I have never seen an abscess.

In the further development of the affection the necessity may arise for the employment of stimulants, for which in the beginning there was no indication. Pallor, marked decubitus, conspicuous weakness, slight apathy, with slight fever are the indications for their employment.

If in such cases mild delirium is added; the pulse becomes weak, small, and frequent; and the disease is at its height, that is to say, on the fifth or sixth day, in some severe cases even earlier,—I resort to the hypodermic use of chininum hydrochloricum. I have repeatedly after one injection seen such a conspicuous improvement that I strongly advise this method of treatment, which is *à priori* rational and is justified by my experience; besides, the method is without the slightest danger. The improvement related less to the temperature, as has been previously mentioned (page 533), than to the general condition; it gives one the impression as if the subcutaneous use of quinin lessens the effect of the products of the bacteria which have found their way into the blood current. A complete drop in the temperature and lasting apyrexia I have seen only in very few instances.

The method of using quinin requires special description. As the hydrochlorate of quinin is only soluble in 34 times the amount of water, to inject ½ gram (7½ grains)—a quantity I have exclusively used in adults—necessitates the injection of 17 gm. (about fʒss) of fluid. For this purpose I have used a modified Koch syringe the glass barrel of which holds at least 10 gm. (say fʒiiss); into its cannula I have had a stop-cock inserted and the bulb is perforated at the side (compare Fig. 31). With this arrangement 10 grams (say, fʒiiss) of fluid may be drawn into the barrel of the syringe, with the stop-cock open, and injected into the subcutaneous tissue; the stop-cock is then closed to prevent the fluid from regurgitating from the tissues into the syringe, and, without removing the instrument, the bulb may be unscrewed, the remaining 7 grams (about f℥ij) introduced into the barrel, and injected at the same point.

I advise as the point of injection the lateral parts of the abdomen. Nowhere is the cellular tissue so loose and so well adapted to take up large quantities of fluid; in no part is the sensibility to pain so slight during and after the injection as at this point. A further advantage is the fact that we may introduce the cannula vertically while the patient is in a horizontal position in bed, so that the glass cylinder also retains a vertical position, which makes the subsequent filling of the tube quite easy.

It is not necessary to previously warm the material which is to be used for the injection if it has been kept in a warm room. If this has not been the case, it is well to previously warm the fluid in a bottle. The skin which is raised at the point of injection is often quite cool to the touch; I have seen no deleterious results follow this.

A further circumstance which requires notice is the cleansing of the syringe. If one rinse the needle and the syringe with a 5% solution of carbolic acid before and after every application, the boiling of the needle will not be necessary. One caution, however, must be employed: One dare not, immediately after having rinsed the syringe with a carbolic acid solution, draw up the quinin solution into the syringe, else, according to the investigation of the chief apothecary of the Altstadt Hospital, Mr. Rosse, there will be deposited crystals of phenol-chininhydrochlorid. The syringe, after having been cleaned with carbolic acid, must be rinsed with a solution of boric acid.

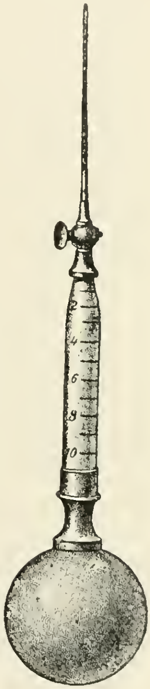

FIG. 31.

In most cases I have obtained favorable results with two injections upon successive days, or with an interval of forty-eight hours between the injections. Frequently one injection was sufficient. Up to the present time I have but twice required 3 injections. The one instance occurred in a severe pneumonia involving the entire right side which was complicated by delirium tremens, so that several doses of chloral hydrate became necessary at night. The quinin injections were used to combat the pneumonia itself, and were therefore given on the sixth, seventh, and ninth days of the disease. The crisis occurred during the night between the tenth and eleventh days of the disease. The patient, a workman aged thirty-eight, was discharged cured. The second instance was a pneumonia occurring after delivery; the result was also favorable.

The value of these quinin injections cannot be better illustrated than by referring to the adjoining table (page 542), statistically arranged in reference to the mortality from pneumonia in the cases treated by me in the hospital during the last eighteen years.

THERE WERE TREATED IN:	MEN.	WOMEN.	TOTAL.	FATAL CASES.			PERCENTAGE.
				Men.	Women.	Total.	
1880	74	7	81	8	—	8	9.8
1881	74	14	88	7	3	10	11.3
1882	67	13	80	13	1	14	17.5
1883	75	17	92	17	3	20	21.7
1884	66	10	76	6	3	9	12.0
1885–86	65	14	79	20	5	25	25.3
1886–87	101	15	116	20	2	22	19.0
1887–88	126	12	138	26	2	28	20.3
1888–89	63	18	81	13	3	16	19.7
1889–90	83	15	98	17	5	22	24.4
1890–91	123	25	148	15	4	19	13.0
1891–92	86	17	103	13	5	18	17.4
1892–93	118	31	149	14	4	18	12.1
1893–94	35	28	63	4	4	8	12.6
1894–95	34	15	49	7	5	12	24.9
1895–96	33	27	60	2	2	4	6.6
1896–97	41	20	61	4	1	5	8.2

The results of the last two years, during which time I employed these quinin injections, speak for themselves. There is no need of specifying the causes of death, such as age, complications, etc., in the fatal cases that occurred during this period; I shall merely add that in the treatment of the four patients who died in the year 1895–96, hypodermic injections of quinin were used only once; and that of the five patients who died during the year 1896–97, three were brought to the hospital in a hopeless condition and died on the day of their admission.

Another useful measure that I formerly employed in the treatment of pneumonia, but have not had occasion to use in the last three years, consists in the employment of diaphoretic agents. When the temperature is very high, while the skin of the trunk is burning hot, the extremities are cold, the pulse small and frequent, and the patient easily falls into collapse with a somewhat clouded intellect, I have had very excellent results with diaphoretics, and recommend their employment in conjunction with quinin injections. But not pilocarpin; that I reject, on account of my previous experiences. My diaphoretic remedy is a much more simple one; it consists in a cup of elder tea to which are added a small liqueur glass of cognac and the juice of half a lemon.

In addition to these measures, which are employed in the treatment of the affection as a whole, I must refer to a symptom on the part of the lungs which also requires attention—namely, the prune-juice expectoration. Patients who expectorate prune-juice sputum do not give a favorable prognosis, as a rule. It is not, of course, to be assumed that the entire condition of the patient can be improved merely by treating this one symptom and possibly causing the thin fluid character of the sputum and the amount of blood contained in it to disappear. But it appears to me that, in addition to stimulants, by administering acetate of lead, which has been recommended by Traube, a favorable influence on this symptom and on the entire disease is produced. I order in such cases 5 cg. (about 1 grain) of acetate of lead with 5 dcg. (say 10 grains) of powdered sugar, preferably in capsules, three times daily. During this period the powders containing quinin and iron are omitted. It is not necessary to employ the actetate of lead longer than two days. I should not hesitate, however, after my present experiences to give the acetate of lead 5 times daily and to use it for three days at a time.

If I say that after the crisis, and occasionally during the period of convalescence, I continue giving the ferrum oxydatum saccharatum or another preparation of iron, nothing remains for me but to speak of certain important symptoms and complications derived from other organs that are involved in the morbid process.

In regard to the *heart*, I have already said that a previous disease of this organ may necessitate the employment of digitalis.

I cannot agree with v. Jürgensen that the fever is responsible for the weakened condition of the heart. This also explains the slight effect of cold baths upon the heart, which are supposed to act by diminishing the fever. If, however, I am in favor of the use of quinin, it is not because it has an effect upon the temperature, but because it diminishes the action of the products of the bacteria which circulate in the blood and produce their effects by weakening the power of the heart. Implication of the endocardium or pericardium requires the proper treatment recommended for such conditions.

The symptoms and complications which arise and are referable to the *brain* require special treatment, as they frequently prove fatal to life. I mention here, above all, the furious delirium, which I believe to be due to an encephalitis (compare page 493). For this I have employed chloral hydrate in doses of at least 3 gm. (45 grains) with excellent effect. The same treatment is effective for the delirium tremens which occurs as a complication of pneumonia. In robust men, even when the pulse has not been especially good, I have employed doses of 4 gm. (ʒj) at a time, and have seen excellent results not only as regards the delirium tremens, but in the general condition of the patient. On several successive nights I have had occasion to use this remedy, and have always seen similarly good results. When meningitis occurs as a complication, I use, as I always do in treating this disease, 5 dcg. (7½ grains) of quinin internally, and at night, and even in the morning, morphin hypodermically. With a protracted course, low fever, cloudy sensorium, small frequent pulse, I advise a daily hot bath at a temperature of 40° C. (104.° F.).

Pleural effusions, even of very large size, usually run a favorable course, be they of a serous or even of a purulent nature, provided proper treatment is instituted. In the former instance salicylic acid in the manner advised by me is used; in the latter case it is absolutely necessary to resort to resection of the ribs. The earlier this is done, the more favorable the results.

Finally, a word in reference to the treatment of pneumonia in women who during the course of pneumonia have given birth to a child. According to clinical experience, and especially according to experimental researches, there is great fear of the development of a puerperal endometritis. If the process is allowed to run its course, the above-described lochial discharge may become offensive, and there is then more danger from the affected uterus than from the diseased lung. In such cases I strongly advise a uterine douche with a warm 2% solution of carbolic acid immediately after delivery; if there is high fever, the douche should be given daily, or even twice a day if necessary. In 2 cases I have by this means,—at that time without the help of quinin injections,—after two or three douchings of the uterus seen very excellent results.

It is hardly necessary to mention that if the tongue becomes dry, as well as in other acute febrile diseases, it should be painted with pure glycerin; if necessary, twice daily.

It is hardly necessary to give minute directions as to diet. At the height of the disease the patients can only take fluid nourishment (milk, cacao, bouillon, soup), and even then they have to be coaxed to eat; stewed fruit, apple-sauce, and oranges may be allowed. Fluid is asked for (water, seltzer water, lemonade); these may be allowed as frequently as the patients ask for them, but not in large amounts, and always provided there be no diarrhea. Toward the end of the disease, especially if it is protracted and during convalescence, everything that may be included under the expression "nourishing diet" should be freely given.

Nursing, the selection of the room, heating, everything is to be taken into consideration, as every detail counts in the endeavor to cure.

[No treatment of pneumonia can be rational without a true conception of the morbid process. It must be understood that we have a pneumococcus infection; that in consequence of this infection we have an inflammation and a toxemia; that the infection may not involve the lungs alone, but other tissues in addition; that if the lungs are extensively involved, one—although the least frequent—of the dangers of the process is dilatation of the right heart; that the toxemia is often out of proportion to the extent of inflammation. Death is due most frequently to the toxemia; next, to associate inflammation in other organs than the lungs; next, to heart embarrassment from the cause above mentioned or from toxemia. It is evident our treatment must be directed to combat the toxemia. This must be done either by neutralizing the toxin, as we do in diphtheria, by limiting its manufacture, by aiding in its elimination, and by antagonizing its poisonous effects with medicine and antidotes. To neutralize the toxins, *anti-*

pneumococcus serum is employed. We have used it in about twenty cases, not hitherto recorded. While we thought we could see benefit in many cases, the number is too small to draw conclusions. This may be said, that my colleagues to whom the cases belonged were pleased with the results in most cases, and all of them will continue to use the serum. No harm attended the employment of the remedy in any instance. The following table gives the more recent observations. It does not seem to support the therapeutic qualities claimed for the remedy. Certainly neither the study of the cases nor the remarks of the authors concerning them bring overwhelming conviction. The number of cases are too few for the percentage of cures to be convincing.

TABLE OF CASES OF PNEUMONIA TREATED WITH ANTIPNEUMOCOCCUS SERUM.

Reporter.	Number of Cases.	Recovered.	Died.	Mortality.
Pane	23	21	2	8.7—%
De Renzi	32	29	3	9.4—%
Lara	10	10	0	0
Bozzolo	5	4	1	20.0%
Massalongo and Franchini	10	7	3	30.0%
Fanoni	6	5	1	16.6+%
Washbourn	6	6	0	0.
Spurrell	1	0	1	100.0%
Cooke	2	2	0	0
Harnett	1	1	0	0
Weisbecker	21	19	2	9.5+%
Maragliano	5	5	0	0
Caruso and Staginetta	2	2	0	0
Gamba	2	2	0	
Marone	1	1	0	0
Cantieri	19	17	2	10.5+%
Canby	4	4	0	0
Wilson	18	14	4	22.2+%
Wilson and Rosenthal	1	0	1	100.0%
Wilson and Page	18	12	6	33.3+%
Kelly	1	0	1	100.0%
Tyler	6	5	1	16.6+%
Rochester	1	1	0	0
Snively	6	5	1	16.6+%
Little	1	1	0	0
Rosenthal	9	7	2	22.2+%
Jones	14	13	1	7.1+%
Bosley	1	1	0	0
Bunte	2	2	0	0
Smith	1	1	0	0
Goldsbrough	4	4	0	0
Crandall	3	2	1	33.3+%
Vandeboncoeur	17	17	0	0
Lambert	12	9	3	25.0%
Total	265	229	36	13.5+%

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To limit the manufacture of the toxins it seems necessary (1) to disinfect parts of the body, as the mouth, nares, and fauces, in which the causal micro-organism has its habitation in health; (2) to allay local inflammation.

1. The mouth, nose, and throat should be washed out with antiseptic washes. The mouth and teeth should be cleansed and kept clean. Ten grains of calomel at the onset, dry on the tongue and allowed to be absorbed slowly, theoretically, at least, is of advantage as an antiseptic. In addition, this drug is of use because of gastric conditions, the mild catarrhal gastritis, perhaps toxic in origin; and because, by its purgative effect, tension is lowered and absorption from the stomach promoted.

2. To allay local pulmonary inflammation, to prevent extension, and to relieve collateral congestion, we employ dry cups, not once daily, but twice or three times a day, if after the first cupping the symptoms are not relieved. Then we employ hydriatric measures, particularly as we can see a threefold indication for their use—first, to allay inflammation; second, to assist elimination; third, to counteract the bad effects of the poison, as, for instance, cardiac paralysis. In addition to hydriatric measures advocated by the distinguished author, we have great confidence in the intermittent application of cold water to the chest, as suggested by Baruch. Several thicknesses of muslin should be cut to fit the anterior and posterior parts of the

chest respectively, the edges overlapping in the axilla. The respective portions, held together by basting, are wrung out of water at a temperature of 60° F. and applied to the parts of the chest they are designed for. They are then covered with several thicknesses of muslin or of flannel, firmly secured with safety-pins, and allowed to remain thirty minutes. In severe cases they should be changed every half-hour or hour; in others, every two hours. If the temperature falls, or the compresses remain cold, they are not reapplied. If reaction results and they get hot, after the lapse of thirty minutes, at the longest, they are removed and reapplied. The object aimed at is to secure deeper respirations, lessen the cyanosis, retard the heart's action, and induce sleep, or at least rest. The compress stimulates the peripheral nerve-endings and reflexly the nervous mechanism of heart and vessels and of respiration. Thus inflammation is mitigated, elimination is hastened. We advocate, with the author, cold sponging, and even the tub, unmindful of the lay prejudice against "cold" in pneumonia. Further to control inflammation, a laxative to lower arterial pressure may be employed. Hence another reason for giving calomel, which, if followed by a saline, lowers blood pressure. Aconite, veratrum viride, and antimony we have never used.

To aid elimination, we employ, in addition to hydrotherapy, agents that stimulate renal secretion; that, in fact, encourage all excretion. Hence we use hot salt or normal serum solutions by the bowel (ʒj salt to Oj water) or by hypodermoclysis—salt solution injected subcutaneously; a pint or more, twice or three times daily. Henry, Osler, and many others testify strongly to the merits of this procedure. We always use it, observing the usual precautions. Caffein, cocain, oil of camphor, nitroglycerin, and strychnin are the medicines which aid elimination. Fortunately, remedies which aid elimination also are, as far as we know, physiologically antagonistic to the toxin, especially strychnin and alcohol. They certainly mitigate the depressing effects of the poison.

To recapitulate: In an uncomplicated case of pneumonia of moderate severity we order mouth disinfection, a dose of calomel, dry cups, and local hydriatric measures. A mild saline fever mixture may be used. One-sixtieth of a grain of strychnin is given every four hours. Indications for the use of caffein, adrenal chlorid, nitroglycerin, digitalis, and cocain are looked for in the heart, the tension of the vessels, and the kidneys. If the renal secretion lessens, if the toxemia is increasing, and the pulse is more rapid and empty, a subcutaneous saline injection is ordered, to be repeated every eight hours. Stimulation is carefully, cautiously, begun. If the right heart is embarrassed, local blood-letting or general venesection is resorted to. In robust subjects with extreme sthenic symptoms general venesection may give great relief. We can strongly indorse the use of iron in pneumococcus septicemia.—Ed.]

CATARRHAL PNEUMONIA.

The credit of having definitely separated lobular catarrhal pneumonia from lobar croupous pneumonia belongs to Rilliet and Barthez. They especially emphasized the fact that lobular pneumonia, even when it has become diffuse,—that is, when a large portion of a lobe is implicated,—still must be differentiated from lobar pneumonia, and that this was entirely possible. When the latter occurs at the base, the process extends toward the apex, and while the base is in the second stage of inflammation, the upper portions of the lung are in the primary stage, and so on, whereas in the case of lobular pneumonia several individual lobules are inflamed and finally unite.

Rautenberg recently attempted to destroy this definite division: I believe, however, unsuccessfully. He states the following theses: (1) The conception of a croupous pneumonia and a catarrhal variety in infancy does not correspond exactly with the microscopic analysis, the macroscopic postmortem appearance, or the clinical course. (2) To designate lobar pneumonia as croupous, and lobular pneumonia as catarrhal, is incorrect. (3) One may speak of larger or smaller pneumonic areas, but not of a lobar or lobular pneumonia. (4) Inflammation of the lung depends upon a disturbance of the tissue which is as yet not sufficiently or definitely investigated, which gives rise to the exit of leucocytes from the blood-vessels into the alveoli of the lungs, and which leads to further metamorphosis. (5) The admixture of fibrin is neither definite nor characteristic. (6) In the classification of pneumonias most weight must be placed upon the etiology.

In all these sentences there is but one that is unassailable; I mean the uselessness of dividing pneumonia into a lobar and a lobular variety. This has also been emphasized by me in a previous section (page 381). The attempt to prove the previously held views of croupous and catarrhal pneumonia as incorrect may be called a failure. In this attempt the exact differential points have been entirely too much neglected. As regards the etiology, the various bacterial causes have not been taken into consideration at all; and as regards the pathology, apart from the investigations which I have submitted, and the exact microscopic findings which I shall report further on, it may be stated that catarrhal pneumonia never has a granular cut surface, and, finally, the clinical course, despite many diagnostic difficulties in the case of children, is sufficiently characteristic to make this division justifiable and possible.

THE PATHOGENESIS OF CATARRHAL PNEUMONIA.

We owe to the exact investigations of Bartels important facts in reference to the cause of the affection. In connection with Charles

West, but upon a sounder basis, he determined that the constant cause of changes in the lungs was always a preceding disease of the bronchi; he, however, came to the conclusion that the disease of the lung is always introduced by collapse of the pulmonary tissue, and that collapse is the first stage in the development of the process within the pulmonary tissue. The further changes he explains by a disturbance of the circulation in the collateral parts. Here an increase in blood pressure occurs, a collateral fluxion with hyperemia gives rise to a transudation of serous fluid in the alveoli, which is followed by disturbances of nutrition in the cells which line these parts (parenchymatous inflammation). Under some circumstances the interstitial tissue also takes part in this nutritive irritation.

No special increase in our anatomic knowledge can be ascribed to the investigations of Steiner, as I must remark, in opposition to other communications relating to this subject. He only arrives at the result that the insular form of thickening of the parenchyma of the lungs, which is characteristic of this process, shows various anatomic varieties. Sometimes this thickening depends alone upon hyperemia of the pulmonary tissue; at other times it is a product of a real inflammation, with either a serous albuminous or a croupous exudate. In other cases the true cause of the thickening is in an excessive proliferation of the nucleus, which may be either intravesicular or extravesicular.

The changes in catarrhal pneumonia are described very minutely by v. Ziemssen. As a constant change an intense catarrhal inflammation of the bronchial mucous membrane is seen; somewhat less in the trachea and the large bronchi than in the smaller and smallest bronchi. After existing for some time a cylindric dilatation takes place. The most constant consecutive change in the parenchyma of the lung is simple atelectasis which shows itself first at the posterior border of both lower lobes. This inflammatory disturbance of nutrition, which has been designated catarrhal pneumonia, begins in the collapsed portions of the lungs. The withered and flabby consistence of these parts disappears gradually. They soon give the sensation of greater resistance, and, on the whole, increase in volume; the finger now plainly differentiates distinct, separate, coarse thickenings of irregular shape and size. On inflating the lung it is noted that the consolidated areas themselves between these areas of atelectatic tissue are not changed by the inflation. They contain in their center an enlarged bronchiole filled with tough secretion. In the further course, by the confluence of many small consolidated foci, large infiltrated areas occur. Fibrinous exudates, such as occur in croupous pneumonia, neither v. Zeimssen nor Bartels were able to find. The pathogenesis, then, may be conceived to be that atelectasis is followed by parenchymatous inflammation, which, if it runs a chronic course, leads to the formation of connective tissue, obliteration of the alveoli, retraction of the affected pulmonary area, and permanent dilatation of the bronchi. It is, however, also specially emphasized that, in addition to the possibility of proving that the chronic form of catarrhal pneumonia develops from pulmonary collapse, the development of isolated inflammatory foci, running an acute course, is not invariably, albeit frequently, preceded by atelectasis. The intense inflammation of the bronchial mucous membrane in acute cases, as it appears, directly spreads to the alveolar wall, and there produces conditions exactly like those that occur in mucous membranes in the form of acute catarrhal inflammation.

The special form of catarrhal pneumonia in which intra vitam and postmortem an inflammatory consolidation of the pulmonary tissue in the shape of striation exists has been described by Steffen under the term striation pneumonia. This streak is found upon one or both sides of the posterior surface of the lungs, reaches in its breadth from the vertebral column to the angle, and either takes in the entire height of one or both lungs from the apex to the base, or affects only certain lobes or even a single lobe, and then is more marked in one or both lower lobes. The cause of striation pneumonia in general is supposed to be poor nourishment and imperfect development in childhood. Rachitis, hereditary syphilis, intestinal processes of long duration, give a proper basis for the development of this affection. In the majority of cases an inflammation of the finer and finest bronchi precedes the affection. The peribronchial tissue is affected in two different ways: Either, as the result of a high grade of swelling of the bronchial mucous membrane and the accumulation of secretion it is made impossible for the air to reach the alveoli, and, as a result, atelectasis occurs; if this should last several days without having been removed by the removal of the secretion, inflammation of the peribronchial tissue develops. Or the inflammation of the bronchial walls occurs directly in the surrounding tissue, without an intermediate stage of atelectasis.

Damaschino, as well as v. Ziemssen, lays stress upon the fact that the bronchitis chiefly attacks the finer and finest bronchi and leads to marked injection of the vascular plexus in the mucous membrane, which becomes intensely red. This reddening by no means depends upon a hemorrhagic imbibition of the mucous membrane or upon an overfilling of the capillary net of the pulmonary artery, which on account of the delicacy of the bronchial mucous membrane merely shows through. In the examination of recent as well as hardened specimens, it is evident that the capillary net of the mucous membrane is actually injected. As a rule, the bronchi in their entire extent are filled with mucopurulent masses, which entirely occlude the opening of the smaller tubes. In the cut surface of such lungs this mucopurulent mass shows itself in the form of greenish drops which remind us somewhat of small abscesses. On lateral pressure small drops that look like pus appear upon the cut surface.

Upon the basis of these explanations and in view of the unanimity of all the authors, we may refer the production of catarrhal pneumonia to a catarrh of the finer bronchi.

My own microscopic investigations also entitle me to say that a catarrhal inflammation of the mucous membrane of the finer and finest bronchi is an absolute condition upon which catarrhal pneumonia depends. In microscopic pictures such as are shown in Plate 5, figures 6 and 7, it is impossible to assume a pure hyperemia. The distention of the capillaries with blood-corpuscles, which persists after death, alone suffices to show the inflammatory character of the changes produced in the tissues, which makes it impossible for the capillaries to empty themselves even in sections taken from the cadaver and hardened. That proper hardening and staining material must be used so as not to cloud the characteristic picture is obvious. I mention here briefly that hardening in bichromate of potassium and staining with Biondi-Heidenhain mixture, as has been previously mentioned (p. 396), have proved exceedingly useful to me.

I am, it is true, merely expressing a personal opinion, although based on numerous comparative observations, when I assert that the engorgement of the smaller bronchial arteries, as represented on Plate 5, figures 6 and 7, is a proof of the inflammatory nature of the changes in the mucous membrane, and that the inability of the tissue and of the vessel wall to force the last remaining blood-corpuscles into the veins during the agonal period, when the heart is ceasing to beat, indicates an inflammatory condition of the tissue and similar changes in the vessel wall. So much is certain: the finding of turgescent bronchial vessels reaching into the capillary system absolutely confirms the diagnosis "catarrhal pneumonia." I have never missed this occurrence in any case of catarrhal pneumonia. I even go so far as to exclude from catarrhal pneumonia deglutition pneumonia, which up to this time has been included, because in this process such changes in the vascular system of the bronchial tree are not present.

If I assert that the condition of the above-described vessels of the bronchial mucous membrane in catarrhal pneumonia is a proof of the inflammatory change, I do so only to demonstrate clearly the meaning of such conditions, not because other proofs are wanting.

In the greatest majority of cases the overfilling of the smaller and finest bronchial vessels, or rather the inflammation of the bronchial mucous membrane characterized by these changes, is accompanied by an exudation into the lumen of the bronchioles which is to be regarded solely as the result of this inflammation. Here the epithelium lies loosened from the walls, as shown in Plate 5, figures 6 and 7, either in the lumen or it is surrounded, where it lies against the wall, by red blood-corpuscles. Besides this, the lumen is almost completely filled with round cells, and in some cases numerous red blood-corpuscles are mixed with them. [The leucocytes infiltrate the walls of the alveoli but do not enter the alveoli, as they are already filled with mucus, cells, and bacteria (Moisseiev).—Ed.] The highest grades of such an admixture, depending upon hemorrhage, I have noted as the result of catarrhal pneumonia following measles. Here hemorrhages occur not only into the lumen of the bronchi, but also in their surroundings and in the neighborhood of the blood-vessels.

According to these findings, the filling and occlusion of the smallest bronchi must be referred to a local exudation from the inflamed mucous membrane. [Moisseiev* states that in catarrhal pneumonia the hepatization in the early stage is due not only to exudation into the alveoli, but to a greater degree to the production of large amounts of mucus by the bacteria themselves.—Ed.]

Besides this, there is still another change: The dilatation of the smallest bronchi, which, according to the opinion of v. Ziemssen, in cases of long duration, constantly occurs in the inflammation of the finer bronchi and frequently reaches a colossal development. The dilatation of the canal conspicuously contrasts with the narrowness of the opening that leads into it, which has retained its normal lumen, or has even become somewhat narrowed on account of the swelling of the mucous membrane. Besides the dark, bluish-red color of the mucous membrane, the bronchial wall as a whole was rigid and thickened, so that upon section through the parenchyma of the lungs, the bronchial stumps rose above the cut surface in the form of yellowish-white nodules.

Steiner also mentions that in a longer duration of the affection the lumina of the bronchi in the posterior and lower parts of the lungs were dilated either in a saccular or a cylindric manner; in one case the dilatation had reached such a degree that the cut surface of the lung had a "coarse-sievelike appearance."

The observations in reference to dilatation of the smaller bronchi I can confirm. The production of this dilatation I refer to the circumstance that the plentiful elastic tissue contained in the parenchyma of the lung is counterbalanced, under normal circumstances, by the elastic tone of the smooth muscular fibers in the bronchial wall; when inflamed, however, the bronchial wall, whose smooth muscular fibers are also disturbed in their function, becomes loosened by the inflammation; it yields to the centrifugal pull of the elastic tissue in the parenchyma of the lung, and thus becomes dilated. The more slowly the inflammation spreads from the bronchial wall to the surrounding pulmonary tissue, the more readily this dilatation occurs.

To these changes in the bronchial wall and in the bronchial lumen those of the parenchyma of the lung are added. Their variety makes an exact description of their processes very difficult.

In the first place, the mechanical results of the occlusion of the smallest bronchi manifest themselves in the pulmonary tissue. The inspiratory power of the thoracic wall and of the diaphragm, especially during childhood, cannot overcome this obstacle in the bronchioles, and the inspiratory air-current is unable to remove the exudate of round cells and the red blood-corpuscles and desquamated epithelial cells from the narrow lumen of the bronchiole. The alveoli which belong to the occluded bronchiole are cut off from communication with the external air. The air contained in them is at least partly absorbed by the capillary vessels of the alveolar wall (Virchow-Bartels); collapse follows in the alveoli of the lungs. The latter collapse, or, as we say, become atelectatic, and the collapsed portions of the lung are recognized at a glance by their reddish-blue, violet, or steel-blue color. The upper surface of these collapsed pulmonary areas is depressed below the level of the surrounding normal parenchyma, and its volume, especially when the entire lobe or the greater part of it has become atelectatic, is greatly diminished. It feels flabby and withered, and no crepitation occurs upon pressure. The distensibility by inflation—the important criterion when it is necessary to differentiate atelectasis from pneumonia—varies according to the duration of the collapse. If the specimen is inflated soon after the occurrence of atelectasis, the normal condition is readily restored. The dark red cut surface, from which but a little blood without air-bubbles could be expressed, after inflation shows itself as light red, and the normal volume is once more apparent. If, however, the atelectasis has been present for a longer time, the hyperemia of the tissue is increased. Upon the cut surface a bloody serum may be expressed. Insufflation in such a case also restores the normal volume, although it requires decidedly more force. But the color of this inflated tissue in cross-sections now appears more scarlet-red, a proof that intense hyperemia had existed. In those cases, finally, in which the atelectasis has existed for a long time and the secretion in the bronchioles has become inspissated, insufflation requires a powerful expiratory effort, and even then the distention of the collapsed part is not complete. Some lobules remain airless, resistant, show upon their cut surface a glistening smooth surface, and in their center constantly a dilated bronchiole filled with inspissated secretion (v. Ziemssen).

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* *Bolnitsch Gaz. Botkins. St. Petersb.*, 1900, xl, 888–892.

The atelectatic areas are exclusively found in the two lower lobes. Only the size and number may be greater in one than in the other. Besides this, in some cases they also occur in the anterior borders of the lungs, especially in the lower tag of the left upper lobe, which overlaps the heart.

In not a few cases, when the disease after a longer duration has terminated in death, atelectasis is not very marked. The changes in the lung consist in nodules of various sizes scattered through the pulmonary tissue, with intervening areas of air-containing tissue. These nodules may vary in size from a pea to a bean. These insular foci of lobular pneumonia, when fully developed, are described by Wyss as nodules of varying sizes, from ¼ of a centimeter to 2 or 3 centimeters or more in diameter. The nodules may be circular or irregular; lobulated or arranged in clusters; of firm consistency; and vary greatly in number, from three or four to as many hundreds, the number being, as a rule, inversely proportionate to the size of the nodules. They are found in the normal, or in injected pneumonic tissue; are often most numerous immediately below the surface, above which they usually project a trifle; or may be scattered through the pulmonary tissue.

Occasionally, however, they are so abundant in one or both bases that the impression of a lobar affection is developed. The cut surface then has a pale, spotted appearance; but it never presents the granular appearance so characteristic of croupous pneumonia.

The simultaneous occurrence of atelectatic and infiltrated areas, the non-appearance of the first, and the continuance of the latter, led to the mistaken opinion that the nodules and nodes, which similar to the atelectatic areas are found principally in the bases, arise from the atelectatic areas. From the atelectasis, so it was believed, a parenchymatous inflammation occurs, which, running a chronic course, leads to connective-tissue formation, obliteration of the alveoli, shrinkage of the portion of lung affected, and permanent dilatation of the bronchi. While this process was supposed to be characteristic of the development of the chronic variety of catarrhal pneumonia only, the development of insular inflammatory areas running an acute course in the most, if not all cases, was regarded as a preliminary stage of atelectasis. In the latter case the inflammation of the mucous membrane spreads directly to the alveolar wall and gives rise to processes analogous to those occurring in the mucous membranes in all acute catarrhal inflammations.

Neither is the opinion of Buhl correct (12 Letters, p. 65), that what is called "catarrhal pneumonia" is not pneumonia at all, but a capillary bronchitis, an inflammation of the bronchioles in which the lung takes part through collateral edema, atelectasis, local emphysema, and engorgement, as a result of the secretion which is displaced from the bronchi into the separate alveolar lobes.

Orth has a different opinion. According to his opinion, "the bronchial contents harbors the causes of inflammation so that the inflammatory process starts in the plug that occludes the bronchial lumen. The inflammation spreads by continuity, affects first the *respiratory bronchioles*, next the adjacent alveoli, and then the alveolar tubes and the alveoli nearest the terminal bronchus, and so forth, until finally the entire lobule is involved."

Kromeyer arrives at analogous conclusions. According to his investigation, the disease develops by means of interstitial inflammatory areas around the ends of the bronchi and from the exudation, which depends on these, into the alveolar spaces. The interstitial peribronchial inflammation is not only the cause of the filling of the alveolar spaces, but also the cause of the persistence of this exudate, as the lymph-vessels are partly obliterated, partly limited in their power of resorption by the proliferating connective tissue.

I have examined microscopically a large number of lungs which were the seat of a catarrhal pneumonia, and have come to the following conclusions:

In the bases of the lungs which were permeated by so many numerous, firm, coarse areas of infiltration that macroscopically no atelectatic areas were visible, there was found in the greatest majority of the alveoli of the lungs a condition which I can scarcely describe better than with the name of "alveolar atelectasis." These alveoli were very small. The epithelium was swollen; and among the mononuclear epithelial cells there were large cells, some without nuclei and some polynuclear forms, which I attribute to the coalescence of several cells of alveolar epithelium, as has been fully explained above (p. 396) in the section devoted to croupous pueumonia. In isolated instances red blood-corpuscles were found among these epithelial elements.

Accordingly a substitution of atelectasis for nodular inflammatory areas must be excluded even in cases in which, with a dense permeation of the tissue with nodules, marked atelectatic areas were absent.

The infiltrated areas of catarrhal pneumonia have no direct relation to the alveoli. They are due to the encroachment of the inflammation from the wall of the bronchiole, and through the wall of the same to the surrounding tissue. As a result of the same inflammatory process which gives rise to the changes in the inner wall of the bronchiole, and therefore has as a result the immediate filling of its lumen with round cells, and eventually with red blood-corpuscles, the entire wall of the bronchiole as well as its external surrounding, becomes permeated by round cells and occasionally by red blood-corpuscles. The neighboring alveoli, whose contents may be readily differentiated from the atelectatic alveoli just described, are only secondarily implicated in this process. In the neighborhood of the inflamed bronchioles the alveoli are of normal size and filled with round cells, between which also desquamated alveolar epithelium is found. Red blood-corpuscles are almost never found among them—a circumstance which is noteworthy, on the one hand, as compared with croupous pneumonia, and, on the other hand, as compared with the frequent finding of red blood-cells at the beginning of an inflammation of the bronchioles, as well as in their surroundings in catarrhal pneumonia itself. As to fibrin, which is said by Damaschino, Henoch, Charcot, and Cadet to be present in the alveoli, neither Bartels, nor v. Ziemssen, nor myself have ever seen it. I may refer in this connection to an obvious source of error. As deglutition pneumonia has usually been counted as belonging to catarrhal pneumonia,—even the deglutition pneumonia which occurs in animals after division of the pneumogastric nerve, which was frequently looked upon as an example of catarrhal pneumonia,—I am inclined to think that the changes which arise in the alveoli have been included in these descriptions; for in deglutition pneumonia of man the affected alveoli are mostly and especially filled by a fibrin net. Whether this is sufficient to exclude the appearance of fibrin in the alveoli in catarrhal pneumonia, I must leave an open question; the erroneous finding of fibrin in the alveoli in catarrhal pneumonia is certainly partly explained by it.

Circumscribed disease of the pleura may be looked upon as a concomitant of the pulmonary affection. Subpleural ecchymoses and circumscribed inflammations of the pleura itself are observed. The former are found at the posterior border of the lungs, in the neighborhood of the atelectatic areas, in older cases also at the base, as well as in the anterior parts. Although at first glance they appear to lie in the pleura, it nevertheless shows itself upon vertical section that they affect the outer cortical layer of the pulmonary tissue and that the pleura is only somewhat bulged out. These ecchymoses are especially found in the pneumonias of whooping-cough and measles (v. Ziemssen). The pleura above the inflammatory nodules, provided the latter extend to the upper surface of the lung, appears constantly in a lesser or milder degree inflamed. The injection in both layers of the pleura is most intense, and corresponds in extent exactly to the inflamed consolidation; very frequently a thick exudate resembling coagulated albumin can be detached from the pleura (v. Ziemssen).

The emphysema, which frequently occurs at the apices, especially at the anterior borders of the lungs, may be regarded as a simple vicarious change. As soon as extensive atelectasis occurs in the bases, a diminution in volume in the lungs must occur, which must be replaced by a part of the pulmonary tissue that has not yet lost its capacity for work and into which the air can still enter. The parts which lie nearest the periphery, the borders and apices of the lungs, must do the greater part of the work, because they lie nearest the thoracic wall, and in deep inspiration must fill out a larger space than under normal circumstances; while the more uniform expansion of the remaining portions of the lobes that have escaped the catarrhal pneumonic process, and their movement from above downward and from below upward during inspiration and expiration respectively, prevents the permanent distention of the pulmonary tissue as a whole.

PREDISPOSING CAUSES OF CATARRHAL PNEUMONIA.

Catarrhal pneumonia is almost exclusively an affection of childhood. [The term bronchopneumonia is interchangeable with catarrhal pneumonia by clinicians in this country. Capillary bronchitis is also a term used in the works on diseases of children for bronchopneumonia or catarrhal pneumonia.—Ed.] This opinion may be proved in the first place by a strict limitation of the disease, which is possible only upon the basis of the anatomic definition previously given. Formerly catarrhal pneumonia was often made to include a number of other diseases, which made an exact conception of this affection difficult in the highest degree. This is true, above all, of deglutition pneumonia, which has no similarity with it, either anatomically or etiologically, and, further, of those pulmonary affections which are due to the inhalation of poisonous gases. If these varieties are excluded, I may say that, except for influenza pneumonia, I have never seen characteristic forms of catarrhal pneumonia in adults. Bartels only describes a case of catarrhal pneumonia in an adult woman, who during an attack of measles died from suffocation, and in this case there was not a trace of pulmonary collapse. Both lungs showed a high degree of edema, and at the bases there were several centrally situated areas, the size of a pigeon's egg, which were mostly devoid of air, and friable. He refers the absence of collapse to the great inspiratory powers in adults.

The nutritive disturbances which we describe as scrofula and rickets, especially in the first few years of life, predispose in a marked degree to catarrhal pneumonia. The pneumonias which arise from such causes, quite properly called cachectic by Rilliet and Barthez, occur very frequently in the poorer population. In this hospital (Magdeburg-Altstadt) also they furnish a very considerable contingent of the diseases of children, and in the majority of cases, owing to their unfavorable course, furnish as large a contingent of the mortality as acute gastro-intestinal catarrh of children arising in the first years of life.

Improper nourishment and bad air in the living rooms may contribute to the development of this form of catarrhal pneumonia just as well as to the development of scrofula and rickets, so that these factors of themselves do not have a direct etiologic relation with catarrhal pneumonia. Only a highly developed rachitic disease of the ribs which prohibits the distention of the lower half of the thorax in inspiration, keeping it retracted, I should like to bring in direct connection with catarrhal pneumonia. I have repeatedly had an opportunity of observing it. Catarrhal pneumonia may also arise in connection with catarrh of the bowel of long duration. The cachexia produced by catarrh may be looked upon as the connecting-link between the two. But in regard to these cachectic pneumonias also, it must be emphasized that their frequency varies greatly in different years.

In the better classes such pneumonias are scarcely ever observed. The predisposition is, in fact, rare among such patients. I am inclined to believe—but it is a mere supposition, based on a small number of observations—that in these cases, especially when the disease occurs in children between the fifth and tenth years of life, the constitutional predisposition is to be ascribed to lues on the part of one or the other parent. At least in the few cases observed by me this was the only fact in the history which could be definitely determined.

Next to the general bodily predisposition the local predisposition of the bronchial mucous membrane plays the chief part.

"That the etiology of catarrhal pneumonia is connected with bronchitis" (v. Jürgensen, Wyss) "cannot be determined with certainty. The symptoms cough and fever may be due to a beginning catarrhal pneumonia, although on the first day of the affection objective symptoms which favor catarrhal pneumonia may not be demonstrable in the lungs."

On the other hand, infectious diseases which arise in infancy and are accompanied by a catarrh of the bronchial mucous membrane are important predisposing causes of catarrhal pneumonia. To these diseases belong, above all others, measles and whooping-cough.

The most thorough investigation of the occurrence of catarrhal pneumonia in connection with measles we owe to Bartels. The predisposition, according to his investigation, is greatest in infancy. Of the children affected by measles, under one year old, 19⅓% were attacked by catarrhal pneumonia; in the ages between one and five years, only 13%; and from five to ten years, only 10⅓%. Only one adult, a very poor and debilitated woman forty-six years of age, was attacked by this pulmonary inflammation and died of it. This case has previously been mentioned (p. 557). Bartels ascribes an important influence in the production of catarrhal pneumonia in measles to unfavorable conditions of life, with bad air and poor ventilation in the living rooms. This explains the rarity of the complication in patients belonging to the well-educated and well-to-do classes.

As regards the stage of measles which is most likely to be complicated with pneumonia, there are, according to Bartels, great variations. Rarely does the pneumonia develop during the acme of the exanthem; in the majority of the cases the eruption had begun to fade or was entirely faded, the fever had already declined, the appetite had returned, and the children had practically convalesced, only suffering from decided bronchial catarrh, when, upon the seventh or eighth day after the appearance of the eruption fever phenomena again appeared, accompanied immediately by marked dyspnea, announcing the onset of the pernicious pulmonary complication. In some cases this occurrence takes place much later, so that weeks may elapse after the disappearance of the eruption, and the little patients, in spite of their catarrh, may be running about in the open air.

That catarrhal pneumonia may arise first, and in the course of the affection the exanthem of measles appear upon the skin, without the possibility of a doubt that the former is an accompaniment of the measles, I once had the opportunity of observing. Perhaps this condition has occurred more frequently, but from lack of sufficient proofs the etiologic relation was not established.

Among my observations there is the case of a boy aged nine, seen upon the sixth of November, 1896. For eight days there had been severe catarrh of the bowel, some fever, cough, and coryza. Upon the ninth an eruption of measles occurred. The course of the disease was very favorable. After two days the fever had disappeared. His sister, aged eleven, developed fever and cough on the 4th of November. These symptoms lasted eighteen days, the fever rising to 40° C. (104° F.). Examination showed a well-developed catarrhal pneumonia of both bases. The characteristic eruption, however, did not appear until the 22d of November, eighteen days after the onset of the catarrhal pneumonia. The fever lasted, under gradual decline of the temperature, for five days. Convalescence then occurred, and the patient was completely restored to health.

As opposed to my view, that in this instance the catarrhal pneumonia was an accompaniment of the measles affection, although preceding it, there is but one other possible view: that the catarrhal pneumonia was an independent affection, not having anything in common with the former disease. This is scarcely justifiable.

From this observation many previous cases of catarrhal pneumonia occurring in children are explained, in which the children came in contact with measles patients, but escaped the measles and only contracted pneumonia. I believe myself justified in concluding from this that there is also a rubeolar catarrhal pneumonia occurring without exanthem.

The occurrence of a catarrhal pneumonia after whooping-cough I believe to be rare, and the conversion of such a pneumonia into tuberculosis has not been proved. The latter I believe to be true, first, because I have had an opportunity of observing that pulmonary affections in children which, upon the ground of the objective examination, were looked upon as tuberculosis, ran their course for weeks and months under the symptom-complex of whooping-cough. Further, I have noted in children who were for weeks and months under my care on account of a cachectic catarrhal pneumonia that after the fatal outcome of the affection no tuberculosis was found at the autopsy. [Shemetschenko* says the specific whooping-cough bacillus (B. Afanasiew) may produce pneumonia, although most of the cases represent a mixed or secondary infection.—Ed.]

More rare, even, catarrhal pneumonia appears to be as the result of diphtheria.

[The most interesting studies of recent years have been by the authors indicated in the following abstract. Councilman, Mallory and Pearce,† after a study of 220 fatal cases of diphtheria, report as follows:

In 131 cases bronchopneumonia of greater or less extent was found; in 76 it was discrete, in 55 it was confluent. In 98 cases it was found associated with diphtheria only; in 33 with diphtheria complicated by either scarlet fever or measles. In the majority of the cases the posterior portion of the lung was affected, especially of the lower lobes; this was particularly true of the confluent form. There was found a definite relation between the presence of membrane in the lower respiratory passages and the occurrence of bronchopneumonia. Thus, of 100 cases with membrane (epiglottis, larynx, trachea, or bronchi), bronchopneumonia was present in 72%; while in the remaining 120 cases it was present in only 48%.

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* *Centralbl. f. Bakteriol.*, Bd. IV, 1888, p. 300.

† *Jour. Boston Soc. Med. Sciences*, vol. V, No. 5.

Of still more interest is the fact that of seventy-six cases which were intubated, 80% had bronchopneumonia. The occurrence of the discrete or the conglomerate form does not appear to bear any relation to the variety of micro-organisms. Nor does the presence of membrane in the bronchi influence the extent of solidification.

The distribution of pneumonia is indicated by the following figures: in 27 cases only one lobe of the lung was involved; in 21, two or more lobes of the lung; in 26, one or more lobes of both lungs. In no case was there a definite lobar pneumonia. The tissue in the vicinity of the solidification was generally edematous, but there was little general edema comparative to that so commonly found in adults: that is, it was edema of inflammation, not general edema due to disturbance of the circulation. When areas of bronchopneumonia joined pleura, the pleural surface was usually slightly cloudy, and in some cases there was evidently a fibrinous exudation confined to small areas.

Histologically, all types of exudates were found,—purulent, fibrinous, hemorrhagic,—sometimes accompanied by necrosis and abscess formation. The type of exudation bore no relation to the character of the infecting micro-organism. Pneumococcus, Streptococcus, Bacillus diphtheriæ, and Staphylococcus pyogenes aureus were commonly found, their frequency being in the order named.—Ed.]

Henoch has called attention to the fact that in such cases there is not only a simple extension of the inflammation from the trachea downward, but that the aspiration of diphtheritic products from the upper air-passages plays an important rôle in this process. Many of the pulmonary inflammations occurring in diphtheria are to be regarded as croupous in character, so that there is but a small part that can be conceded to catarrhal pneumonia. [Streptococcus pyogenes is often the most active agent.—Ed.]

On the other hand, I believe that influenza, in comparison with its frequency, is an especially frequent cause of catarrhal pneumonia, and the most important cause of catarrhal pneumonia in adults. If there were a greater amount of material as regards microscopic examination in my possession, I could with more certainty than now, after the examination of four cases, say that influenza pneumonia is in fact nothing but a variety of catarrhal pneumonia. Of course, I must observe beforehand that such a division may meet with many objections; according to the opinion of a number of very prominent authors, influenzal pneumonia may be any one of a number of different forms of pulmonary inflammation. Rebert reports six fatal cases as a result of influenza, in which the autopsy showed more or less well-developed pulmonary inflammation. In three cases this inflammation took in an entire lobe, and was therefore a lobar affection, but these cases differ in many ways from croupous pneumonia. The cut surface of the hepatized lobes was smooth, not granular, the exudate soft, very rich in cells and poor in fibrin. In one case the lobar consolidation of the cut surface was divided in a lobular manner, and the individual lobules were not uniformly changed. In three of the other observations, and partly also in the fourth, the consolidation was composed of separate areas, some presenting a flat, and some a granular, surface on section. Taking into account the reports of other authors, Leichtenstern, in his description of pneumonia, says that as a result of this disease croupous lobar and lobular pneumonias also occur as catarrhal pneumonias. He distinguishes four varieties of influenza pneumonia—the catarrhal bronchopneumonia, the fibrinous, the cellular-fibrinous, and the acute lobar cellular pneumonia, which latter is equivalent to the acute lobar desquamative pneumonia described by Buhl.

Pfeiffer and Beck, however, explain the pathologic picture of the lungs in fatal cases of typical influenza pneumonia as being very characteristic, in so far as no uniform hepatization is found, but more rarely numerous bronchopneumonic areas of variable dimensions, which may partly become confluent and mostly occur at the bases. In the center of such bronchopneumonic areas one frequently notes upon section bronchial twigs, from which a greenish-yellow, purulent, tough secretion can be expressed. Upon microscopic examination these plugs show themselves to be composed of pus cells that are embedded in tough mucus, and one notes in them, partly free and partly embedded in cells, the typical influenza bacilli, often in enormous numbers and frequently in pure culture. This proves that the influenza sputum is produced, at least in part, in the finer ramifications of the bronchial twigs. In the pulmonary substance itself one finds, near the bronchi,—often, however, apparently without any connection with these,—the pneumonic areas, which are readily seen with the naked eye. In the center of these the entire tissue, alveoli, and alveolar septa, as a rule, are filled with typical pus cells, so that the pulmonary structure appears to have entirely disappeared. The surrounding parts of the lung show themselves to be in a condition of desquamative inflammation. The more the large cells which here fill the alveolar lumen are separated from the center of the area, the more sparingly are leucocytes mixed with them. Fibrin in these peculiar pulmonary changes can only rarely be found.

The minute microscopic examination of the four fatal cases of influenza pneumonia occurring in the winter of 1896–97 forced me to revise the opinion based on the macroscopic examination of six cases which came to autopsy during the epidemic of influenza pneumonia in the year 1890. At that time I also inclined to the opinion that, in view of the uniform implication of one or several lobes in rapidly fatal cases, and in view of the almost entire absence of air in the tissue,—in other words, the hepatization,—the process was more like croupous than like catarrhal pneumonia. This view was apparently confirmed by the occurrence of scattered areas which could only with difficulty or not at all be differentiated from the gray hepatization of a croupous pneumonia.

But in spite of the apparently uniform implication of the tissue of an entire pulmonary lobe, the character of the bronchial vessels and the contents of the finest bronchi showed on microscopic examination that the affection in question was a catarrhal pneumonia. The statement in regard to the classification of pulmonary inflammations on page 381,—namely, the uselessness of a division into lobar and lobular inflammation,—applies with especial force to influenza pneumonia. As little as lobar is identical with croupous, just so little is lobular identical with catarrhal; a catarrhal inflammation of the lungs may occur in an entire lobe *in toto*. It may, therefore, be lobar.

In no other variety of catarrhal pneumonia, however, have I seen such uniform implication of one or more lobes as occurs in influenza. As I carefully examined the specimens taken from the before-mentioned cases, which were stained with a Biondi-Heidenhain mixture, I was astounded to find that in regard to the condition of the vessels I could not find the slightest parallel with the condition which occurs in croupous pneumonia. The arteries and capillaries of the finest bronchi were completely filled, the lumen of the finest bronchioles contained desquamated epithelium and numerous white and red blood-corpuscles.

If the definition given by me for catarrhal pneumonia—inflammation of the mucous membrane of the smallest bronchi and inflammatory hyperemia of the bronchial vessels (compare Plate 5, Figs. 6 and 7), which does not occur in croupous pneumonia (compare Plates 4 and 5, Figs. 1 to 5)—be correct, the influenza pneumonia must also be included under catarrhal pneumonia.

I must emphasize only one difference in regard to the overfilling of the vascular system between catarrhal pneumonia arising in the course of influenza and the catarrhal pneumonia occurring from other causes. In influenza there was found the highest grade of hyperemia at the transition of the bronchioles into the alveoli and in the walls of the alveoli themselves, whereas this hyperemia in other cases stops short of the alveoli.

This condition of the vessels best explains the occurrence of gray areas in the uniformly inflamed red basic tissue in influenza pneumonia. These gray areas are unquestionably alveolar inflammatory foci, which are produced by the filling of the alveoli with white blood-corpuscles, without an accompanying fibrinous exudation. Their origin may be explained by the fact that the inflammatory hyperemia of the wall of the terminal bronchi is continued to the walls of the alveoli, and here gives rise to an exudation which is analogous to that which takes place in the smallest bronchi, but must take a shape corresponding to the locality.

It would therefore be superfluous to make the attempt to explain such alveolar pneumonic areas as due to the combined action of the influenza bacillus and the pneumococcus, especially as the attempt to prove the existence of pneumococci in influenza pneumonia cannot be looked upon as having been successful. Wassermann says: "The opinion that influenza produces the field in which the pneumococci and streptococci may develop their pathogenic activity (Ruhemann) would only then be tenable if in the case of influenza pneumonias we always found pneumococci and streptococci. This is, however, not the case; on the contrary, they are almost always absent, so that in pure uncomplicated cases the pneumonic sputum is found to contain only an absolutely pure culture of influenza bacilli. According to this, such a pneumonia—and during an epidemic there are mostly cases of this kind—cannot be looked upon as any other complication of influenza, but simply the extension of one and the same process from the bronchi into the pulmonary tissue. In other words: Influenza pneumonia has nothing in common with genuine croupous pneumonia. It is a variety in itself."

I must add that in the fluid separated from the lung tissue in influenza, in addition to numerous influenza bacilli, diplococci of pneumonia are also found. Does this render the above deductions worthless? From the histologic findings, certainly not.

Influenza pneumonias, although they arise from a catarrhal inflammation, usually assume a lobar form, and very rarely lead to the production of lobular areas, because the affection almost always occurs in adults. In children the exudate which is produced in the bronchioles easily leads to occlusion, and the plugs produce peribronchial inflammation, resulting in lobular areas. In adults, on the other hand, the inspiratory powers may remove the obstruction and keep open the air-passages into the alveoli, thus retarding and sometimes even preventing the production of lobular peribronchial areas, and a diffuse spreading of the inflammation over all terminal bronchi, or, in other words, producing a lobular form of the affection. [In short, influenza may be attended by influenza pneumonia, from the Pfeiffer bacillus, or may invite other pneumonic infections, as from the pneumobacillus, the staphylococcus, or the streptococcus.

Houl[*] considers many bacteria which are causal, as:

Streptococcus pyogenes may be the cause of a primary pneumonia, of an acute lobar pneumonia, though more often of the lobular type. Mosny[†] tells of a case of such a streptococcic pneumonia, derived from contact with a patient suffering from facial erysipelas. As the cause of the secondary pneumonias following the infectious fevers, and of the aspiration pneumonias, the streptococcus is preeminent. Holdken describes a similar infection in four cases in the same house, two fatal; streptococci recovered from blood. Neissmayer reports three cases also, and thinks the duration longer than ordinary in this form of infection.

Staphylococcus pyogenes aureus and albus are often the cause of the secondary pneumonia of the infectious diseases, either alone or more usually in combination with other micro-organisms.

Various micro-organisms have been occasionally described as the cause of single or a few cases of pneumonia. Thus Schau[‡] tells of a

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[*] "Ergebnisse der allgemeinen Aetiologie der Menschen," etc., 1896, i, p. 667.

[†] *La Semaine Méd.*, 1890, No. 7.

[‡] *Fortschritte der Medicin*, 1885, No. 15.

Bacillus pneumonicus agilis, which Neumann[*] found in a case of pneumonia occurring during the course of variola. Klein[†] describes a Bacillus pneumoniæ, probably identical with Friedländer's pneumobacillus. Mosler[‡] found a special bacillus, and Babès[§] describes a Bacillus pneumosepticus isolated from a case of septic pneumonia. The Bacillus enteritidis is made responsible for a case of pneumonia by Lubarsch,[||] and Marchand[**] found a Bacillus capsulatus. Alfieri[††] tells of a special bacillus, which grows as the typhoid bacillus does, but is stained by the Gram method, and produces a foul odor when cultured. Fischer and Levy[‡‡] think the Bacterium coli was the active agent in causing a bronchopneumonia after an operation for a gangrenous hernia. Koch's bacillus is the immediate cause of pneumonia tuberculosa or pneumonia caseosa, though Ortner[§§] considers the disease a mixed infection, in which the pneumococcus prepares the way for the tubercle bacillus, which works upon the already affected lung; *i. e.*, changes the cellular exudate into a cheesy one. Fraenkel and Troje[||||] oppose this view, and consider the pneumonia of tuberculosis as due primarily to the tubercle bacillus. Terray[***] states that in a case in which croupous pneumonia and tuberculosis had been diagnosticated, both Friedländer's pneumobacillus and the tubercle bacillus were found. Section proved the diagnosis to be correct. Bacillus mallei may be the primary cause of pneumonia, reaching the lung with the inspired air or through the circulation. A secondary septic pneumonia may also develop upon the favorable soil found during or after an attack of glanders.

Finally, I must mention as a cause of catarrhal pneumonia in children the action of irritating substances upon the mucous membrane of the bronchi. At least I may bring the continuous inhalation of smoke, which may lead to unconsciousness during a fire, into this category. On the 27th of February, 1897, the wife of the workman L. had a business engagement. She locked her house and left her three perfectly healthy children, two girls aged five and three, and a boy aged six, in the house without any one to take care of them. When she returned after two hours, at eleven o'clock in the morning, the room was full of dense smoke; the children were found lying unconscious upon the floor. A large feather-bed was partly burned and still smoking. The children had found, as frequently occurs, matches, and in this way the accident occurred. Upon the advice of a physician who was called in, both of the elder children were admitted to the hospital in the afternoon; the mother retained the youngest, which died after twenty-four hours. Both of the elder children were still unconscious when admitted. After some cold applications they regained consciousness. The three-year-old girl had marked cough. Her temperature in the evening was 40.3° C. (104.5° F.). The respirations were very frequent—60 in the minute. Percussion of the lungs showed nothing abnormal; auscultation over both lungs showed sibilant and sonorous râles. The next day crepitation could be elicited over the right base. Somewhat later, crepitation was also noted at the left base. While these objective changes as well as the fever persisted the dyspnea continued, and four days after the accident death occurred. The autopsy showed a typical catarrhal pneumonia of both bases and of the middle lobe. In a microscopic examination all of the above-mentioned criteria of these pneumonias were absolutely determined. In the oldest boy, aged five, a catarrhal pneumonia also occurred, which ran its course with very severe symptoms, but terminated in recovery.

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[*] *Zeitschr. f. klin. Med.*, Bd. xii, Heft. 1, 1887, p. 73.

[†] *Centralbl. f. Bakteriol. u. Parasitenkunde*, Bd. v, No. 19, p. 625.

[‡] *Deutsche med. Wochenschr.*, 1889, Nos. 13 and 14.

[§] "Eisenberg-Diagnostik," 283.

[||] *Virchow's Archiv*, vol. cxxiii, p. 70.

[**] *Centralbl. f. Bakteriol. u. Parasitenkunde*, Bd. xv, 1894, p. 428.

[††] *Centralbl. f. Bakteriol. u. Parasitenkunde*, Bd. xvi, 1894, p. 36.

[‡‡] *Centralbl. f. Bakteriol. u. Parasitenkunde*, Bd. xii, 1892, S. 478.

[§§] "Die Lungentuberculose als Mischinfektion," Wien, 1893.

[||||] *Zeitschr. f. klin. Med.*, 1894.

[***] *Centralbl. f. Bakteriol. u. Parasitenkunde*, Bd. xi, 1887, p. 557.

The similarity of the affection in both of the older children, who up to that time had been well, allows us to refer the origin of the pulmonary changes to the inhalation of the smoke. It must remain undecided whether the deleterious effect upon the mucous membrane of the finer bronchi was produced by the smoke itself or by any other product of the smoke which was inhaled. The influence of the latter appears to me to be the more likely; at least I was not able to find any particles of coal-dust in the lungs.—Ed.]

ETIOLOGY OF CATARRHAL PNEUMONIA.

As a direct cause of catarrhal pneumonia we must look upon the action of the bacteria upon the mucous membrane of the smaller bronchi. But the fact that the affection shows no uniform character, in so far as it may be partly primary and partly occur in connection with other, especially acute diseases localized in the mucous membrane of the air-passages,—for example, measles, influenza,—the assumption is tenable that many forms of bacteria may give rise to the disease. The assumption, which is tenable in other affections, that a special variety of bacillus is added to that of the primary affection, as, for example, measles, is negatived in this disease by the fact already referred to that the poison of measles may first produce a catarrhal pneumonia, while the rash need not occur until after the pulmonary affection has existed for some time, or may even be absent entirely.

Cornil and Babès quite properly declare that in bronchopneumonia in general the micro-organism of the various infectious diseases that give rise to the pulmonary affection are found. Netter examined 95 cases of bronchopneumonia—53 in adults, 42 in children. The bacteriologic examination, however, developed four different varieties of bacteria: Pneumococcus, Streptococcus pyogenes, the capsule bacillus of Friedländer, and Staphylococcus pyogenes aureus. As a rule, a bronchopneumonic area only contains one form of these micro-organisms, but several varieties may be noted in the same area, and especially in children. The streptococci have been most frequently found in pneumonias occurring in the course of diphtheria, erysipelas, and puerperal fever.

No other or more exact results have been developed by later investigations [except in the case of diphtheria.—Ed.].

THE SYMPTOMS OF CATARRHAL PNEUMONIA.

Catarrhal pneumonia belongs to those diseases which are characterized by great variability of symptoms, as well as by a duration which may vary within wide limits.

With reference to the latter circumstance, the duration of the affection, an acute and a chronic form may be distinguished. Such a division is, however, without special value. A classification to correspond strictly to the course of the affection, should include only acute and subacute forms, for there are cases which, as regards the symptoms, and especially the continuous high fever, must be designated acute, although they last up to three weeks; whereas the cases which are designated chronic rarely last longer than three months.

This variability in the duration of the affection depends upon the different forms of inflammation previously mentioned. A primary catarrhal pneumonia, arising in previously healthy children, rarely lasts less than a week or longer than three weeks. The variety occurring after measles and whooping-cough may terminate fatally in three days, but may also last for weeks. Cachectic catarrhal pneumonia, arising in scrofulous and rachitic children, lasts from one to three months and usually terminates fatally.

The slowest course, according to v. Ziemssen, is observed in the catarrhal pneumonia which develops after whooping-cough. The antecedent atelectasis, the slow development of the infiltration, the moderate degree of fever, the substantive emphysema of the upper lobes, which almost always complicates these pneumonias, are phenomena which deviate markedly from the picture of the pneumonia occurring in measles.

The character and severity of the symptoms, like the duration, vary in the different forms of the affection. I saw a primary catarrhal pneumonia in a girl aged nine which led to high fever lasting fourteen days, but presented no other subjective symptoms, ran its course almost without cough, and was only demonstrable by dulness and feeble respiration over one of the lower lobes. Recovery took place in this case.

As a rule, however, marked symptoms are present on the part of the respiratory organs, which, besides the fever, plainly characterize the affection.

The fever itself usually rises slowly in the primary form. Inside of three days it may reach a considerable height. Temperatures of over 40° C. (104° F.) are not rare. The fever may persist at this level with only slight morning remissions for eight days; in some few cases even longer; the defervescence is usually slow by lysis.

Catarrhal pneumonia occurring after measles is especially characterized by high fever. If the pneumonia occur during the eruptive period of measles, then it is usual for a marked increase of the fever to occur. If the temperature is neglected, the complication of pneumonia may easily be overlooked. Von Ziemssen, therefore, lays great value upon a thermometric record of the fever, because without this, a well-developed bronchopneumonia or a croupous pneumonia may easily be overlooked. The physical signs are very similar to those of a croupous pneumonia. A markedly developed dulness, which does not necessarily always have to occur over a lower lobe, usually arises at the height of the rash, with other severe symptoms, or during the period of desquamation, within thirty-six to forty-eight hours. Whereas, however, in croupous pneumonia a sudden fall in the fever, with critical symptoms, occurs in the greatest majority of cases, this is almost entirely absent in catarrhal pneumonia. In the latter the temperature comes down gradually, so that the normal temperature is reached on the third or fourth day from the beginning of defervescence, at the earliest.

Unquestionably this point of v. Ziemssen's in reference to the course of the temperature is of great value for diagnosis, but it does not prove the occurrence of catarrhal pneumonia in the apices, which I have never seen even in measles.

A lower temperature is usually observed in the cachectic, catarrhal pneumonias. In fatal cases the fever may occasionally be absent entirely, especially toward the end of the affection. Even subnormal temperatures may occur. Steffen lays great stress upon the sudden development of subnormal temperature in children in the first years of life. He regularly found dulness over the posterior surfaces of the lungs in these cases.

The objective examination of the lungs yields very variable results, which depend in every case upon the duration of the affection, and in an individual case on the number of individual inflammatory areas.

At first, auscultation over the affected areas of the lung only shows the signs of a catarrh—feeble breathing, with moist râles. This, in connection with the febrile conditions, is an important diagnostic point. If, however, only medium-sized moist râles later develop, it will require very careful investigation and the help of all other subjective and objective symptoms, especially the results of percussion, to determine the diagnosis. Only if bronchial breathing occurs in the further course of the affection, a positive diagnostic sign is given by auscultation, but this does not frequently occur. The bronchopneumonic areas must have become very numerous so that they almost coalesce.

Percussion may give rise to mistakes in diagnosis, and this must be especially emphasized. I have repeatedly, in percussion of the posterior surface of the thorax over the apices, found a dull note; whereas, upon the base the percussion note was hollow and slightly tympanitic. As in such cases, at least over the fossæ supraspinatæ, there is at least rough, almost bronchial, breathing, I formerly believed that the catarrhal pneumonia must also have affected the apices. The autopsy—these cases were almost exclusively in cachectic children who usually died—showed me that I was mistaken. The apices, in spite of the results of the physical examination, were normal; the affection occurred exclusively in the bases. These changes in the percussion note over the normal apex can be explained only by the fact that under the influence of the changes at the base the tension of the normal tissue at the apices had been modified, so that the percussion note, as compared with the high tympanitic quality over the lower lobes, appeared empty and almost dull. The error may also be partly due to the difference between the tympanitic note over the lower lobes and the note obtained over the upper lobes, which give forth a percutory note that is but little shorter than the note obtained over a normal lung, so that the latter is apt to be mistaken for dulness.

Such clinical findings may explain the assumption that catarrhal pneumonia in children may also occur at the apices. Of more value and more characteristic is the percussion over the bases in the further course of the affection. As soon as dulness occurs over both lower lobes, or only over one with a shortened or tympanitic percussion note over the other, the previously mentioned predisposing causes, taken in conjunction with the febrile phenomena and the results of auscultation, make up a complete group of symptoms, which suffices to render the diagnosis fairly definite.

But in most cases the results of percussion are not so characteristic. They consist merely in a slightly tympanitic element in the percussion note over the bases. Usually the degree of this note is unequal, if we percuss different areas of the same base; but this in itself is a valuable diagnostic sign. Equally valuable is the fact that in the course of the disease, within a short period, even between one morning and the second, or upon the evening of the same day, an examination will show that the note over the bases changes, that one area which was tympanitic in the morning, in the evening gives an empty high-pitched note.

The increased feeling of resistance also which is noted in percussion of the thorax over the infiltrated areas has had great stress laid upon it by Wyss. This change of resistance is the more easily demonstrated, the more extensive, the thicker, and the more densely infiltrated the pulmonary areas are. The determination of the resistance is of value in all parts of the thorax, but especially in those which are sparingly covered with soft parts; that is, the posterior and lateral surfaces of the chest.

Cough is one of the symptoms that are constantly present. The case quoted above, in which no cough occurred, may be looked upon as extraordinarily rare, and need scarcely be taken into consideration. As a rule, the attacks of cough are brief; but they may occur very frequently. I have never observed any marked changes in the variety—that is, in the intensity and duration of the paroxysms of cough in measles—due to the complication of a catarrhal pneumonia. If whooping-cough precedes the affection, the characteristic paroxysms of cough are said to disappear during the catarrhal pneumonia. I myself have never had occasion to notice such a modification. [The editor has frequently observed this.]

Occasionally there is a simultaneous catarrh of the larynx; *i. e.*, hoarseness and aphonia. This complication may make the diagnosis of the condition very difficult. I shall revert to this.

Sputum is not present in children in the first years of life; it is swallowed. The bronchial secretion in general does not appear to be particularly copious.

Pains are not frequent; they increase still further the respiratory frequency, which is already quite marked. In children in the first years of life 60 and even 80 respirations per minute are not rare. With this, as a rule, there is no irregularity. In cachectic catarrhal pneumonia the respiratory frequency need not be especially increased.

Cyanosis of the skin, especially of the lips, is frequently observed. With an improvement in the disease the respiratory frequency decreases and cyanosis rapidly disappears.

Occasionally marked dyspnea occurs. It is impossible for children to lie upon their backs. They sit up for hours in their beds. Once I saw a strong child one year of age, affected by catarrhal pneumonia, stand the entire day; every attempt to get it to assume the recumbent posture was useless. Only at night would it lie for some hours upon its back while sleeping.

In connection with dyspnea a retraction in the lower parts of the thorax may occur. A preceding rickets may not be without influence upon the frequency of this occurrence; however, this may occur independent of it. Of importance for the origin of this retraction is the distribution of the consolidated areas, as well as atelectasis, at the bases of the lungs. The larger these are, the smaller must be the volume of the entire lower lobe of the lung. In inspiration such a diminished lower lobe must be drawn down by the diaphragm, into a part of the thorax whose frontal and sagittal diameters must increase from above downward. Under normal circumstances this is compensated for by the expansion of the alveoli of the lung, or, in other words, the distended lower portion of the thorax is filled by the lower lobes during inspiration. If, however, such an expansion of the lung is not possible, the retraction at the lower aperture of the thorax must compensate for the increase in the volume of the thoracic space caused by the inspiratory depression of the vault of the diaphragm.

If such a retraction occur with simultaneous hoarseness, then the differentiation from croup, with an existing stenosis of the larynx, and a retraction of the lower parts of the thorax due to this, may become exceedingly difficult, especially in cases which are treated in a late stage of the disease.

Some differences from the symptoms here described are observed in influenza pneumonia, which, in contrast to all other forms of catarrhal pneumonia, occurs in adults. Leyden says [155]: Influenza pneumonia has many peculiarities. In some cases true hepatization does not occur. The congestion of the lungs shows itself by crepitant râles; which appear and disappear rapidly; in a number of cases no real dulness or bronchial breathing occurs; nevertheless these cases run a course similar to that of a regular pneumonia; that is, there is usually high fever up to the sixth, even to the seventh day, when the crisis occurs. The expectoration in some few cases is distinctly rusty; in other cases there is no bloody expectoration, while in a third group of cases a trace of glairy, slightly bloody, or rusty sputum is obtained, which is, however, much less abundant than in the usual typical cases of pneumonia. The course of these cases usually, but not always, coincides with that of the local affection; some few cases terminate fatally without arriving at a well-marked pneumonic infiltration. [Suppression of breath sounds, moist râles, and impaired resonance over an entire lung posteriorly may be the only physical sign.—Ed.]

Among other organs affected by this process in a symptomatic respect the nervous system requires special description. Children are usually very restless. They throw themselves from side to side and are delirious. In a few cases I have seen rigidity of the muscles of the neck; these cases terminated fatally. At the autopsy no abnormality of the brain or of the meninges could be seen macroscopically. Even the fluid of the ventricles could scarcely be said to be increased.

In other cases the children are almost constantly somnolent. As in these cases there is usually cyanosis of the skin, it is obvious that this condition of the sensorium depends principally upon a deficient oxidation of the blood.

COMPLICATIONS AND COURSE.

A complete review of all the literature in reference to the complications and course of catarrhal pneumonia, after the definition of the disease which has here been given, would be exceedingly difficult. The majority of physicians who have given detailed descriptions of catarrhal pneumonia have included deglutition pneumonia as one of the varieties, and laid the sequels of this disease promiscuously at the door of catarrhal pneumonia. In a description in which these two essentially different varieties are treated separately these experiences can only be partially utilized.

The statement that pleurisy is a comparatively frequent complication of catarrhal pneumonia seems to me to be very uncertain. According to my investigations, it is a very rare complication of pneumonia, whereas in deglutition pneumonia I have found it very frequent. In these rare cases the pleural exudate was quite as frequently purulent as it was serous. Following measles, I saw once in a boy aged five an empyema filling nearly the entire left side of the thorax, and by means of resection of the ribs a cure was obtained without deformity of the spinal column. The boy is now fourteen years old and for the past six months has had a slight infiltration of the right apex.

Pneumothorax was seen by Steffen, in his 76 cases of stripe pneumonia, but twice. I cannot, however, look upon these two cases as belonging to catarrhal pneumonia.

Most difficult is the answer to the question as to the frequency of tuberculosis, especially miliary tuberculosis, after catarrhal pneumonia. The opinions of various authors differ markedly in this respect. I quote Wyss, who says: "Miliary tuberculosis is one of the most frequent complications of chronic, especially of caseous catarrhal pneumonia; it often plays the part of a terminal process, and may spread in various ways through the entire organism; most frequently as a local tuberculosis, surrounding the caseous areas in the lung, or as a diffuse pulmonary tuberculosis; or one finds no tubercles at all in the lung, but only the catarrhal pneumonic process, with, however, miliary tuberculosis of the meninges or of the abdominal organs or of both together. Von Ziemssen, however, declares: "The termination in acute or chronic tuberculosis was supposed formerly to occur very frequently after measles or whooping-cough. I must, however, declare this termination to be relatively rare, and can substantiate the reports of Bartels, who in 21 autopsies only twice saw fresh miliary tuberculosis of the lungs, and twice found a similar condition in the meninges."

I must coincide with this latter opinion, and must especially point out that many circumstances have led to the erroneous supposition of the development of tuberculosis from catarrhal pneumonia. In children a primary tuberculous bronchopneumonia may clinically be looked upon as a pure catarrhal pneumonia, because the diagnostic difficulties in these two forms are exceedingly great. If, then, the fatal termination occurs, it is natural to ascribe the miliary tuberculosis found at the autopsy to a primary catarrhal pneumonia.

In other cases a primary tubercular bronchopneumonia in children may symptomatically resemble whooping-cough. The autopsy then leads to the conclusion that the tuberculosis preceded the whooping-cough.

Even then if it be sufficiently proved that measles preceded and that the pulmonary affection was secondary and itself terminated in tuberculosis, there cannot always be found a direct or an indirect causal relation between measles, catarrhal pneumonia, and tuberculosis. This is especially true of cases of measles occurring in cachectic scrofulous children. In this infectious febrile disease tubercle bacilli may be freed from scrofulous or cheesy lymph-glands in the mesentery or mediastinum, and in this manner a general miliary tuberculosis may result.

The termination of the process in a caseous degeneration was looked upon as an exaggeration of the pneumonic process, and this was characterized by the majority of authors as a very frequent event. I myself have not noted a single instance which would justify me in believing that a catarrhal pneumonia had terminated in a caseous degeneration; in the few observations at hand it was impossible for me to exclude a primary tubercular bronchopneumonia upon a hereditary basis. If it was possible for v. Ziemssen to follow during life two cases of the caseous metamorphosis of the inflammatory products for so-called tuberculization, and in one case to demonstrate it at the autopsy, I must in regard to the latter have the same doubts as in my own cases. His case occurred in a "starved, artificially fed" child, which from birth was affected by catarrh of the bronchial and intestinal mucous membranes; it was five months old, and was attacked by a left-sided catarrhal pneumonia. Consolidation was very slow, extending from the base upward, so that it took three months for the entire left lung to become atelectatic. Death occurred four months after the beginning of the pneumonia, after the left lung had for some weeks shown signs of a cavity, and consolidation and atelectasis had also developed on the right side. The autopsy showed extensive tissue-destruction in the left lung, both in the upper and in the lower lobes. The consolidated tissue which remained was partly stained yellow, whereas at other parts newly formed connective tissue could be noted. No special explanation of this latter point is deemed necessary. Since the discovery of the tubercle bacillus there can be no question of a differentiation between cheesy pneumonic exudates and tuberculous disease of the lung. The cause is the same; the anatomic variety may perhaps be dependent upon the ages of those affected.

No less doubtful is the appearance of abscesses in catarrhal pneumonia. They are confounded with collections of pus inside the bronchial lumen which are not infrequently found and, if the bronchus undergoes saccular dilatation, may assume a spherical form. Real abscess arising from destruction of the pulmonary tissue Damaschino had already declared to be exceedingly rare. According to his observation, such abscesses rarely attain a larger size than a nut. The surrounding tissue was inflamed, and in the condition of gray hepatization. Wyss further explains the formation of abscess from a softening in the center of an infiltrated pulmonary lobule. The small punctiform abscesses appear as yellow or gray points, which, when opened, allow a drop of pus to escape. He also speaks of the finding of larger abscesses that have arisen by the decomposition of the pulmonary tissue, from which it may be deduced that in the case of the former the parenchyma of the lung was not affected. Zenker reports the formation of an abscess in a case of croupous pneumonia following influenza. He refers the origin to a diminution in the expiratory power of the lungs due to emphysema, in the presence of enormous numbers of Fraenkel's pneumococci which had acquired unusual virulence through symbiosis with influenza bacilli.

Gangrene of the lung, according to Wyss, occurs in pneumonia following measles and in pneumonia due to the inhalation of foreign bodies. The latter need not be taken into account at this place. In the former, it must belong to the greatest rarities.

The termination in connective-tissue formation, and, finally, cirrhotic contraction of the pulmonary tissue, v. Ziemssen has never seen. Bartels describes a case in which in a nine-year-old girl, following a pneumonia due to measles, the entire left lung gradually developed cirrhosis, affecting nearly the entire left apex, whereas the base returned to its normal condition.

Catarrhal pneumonia is also responsible for the swelling of the bronchial lymphatic glands. This condition may, however, have existed before the catarrhal pneumonia, especially in the numerous cases of cachectic catarrhal pneumonia.

Wyss has seen pericarditis once in connection with catarrhal pneumonia following a chronic bronchitis, being complicated by a left-sided pleurisy in a child aged twenty weeks (pericardial friction was present). He verified his diagnosis at the autopsy. He also demonstrated other cases at the autopsy; among these, a case following catarrhal pneumonia due to measles. Steffen and Steiner have noted similar cases.

Enlargement of the liver and spleen, or one of these organs, I have repeatedly found in catarrhal pneumonia. I believe it is due to a preceding scrofula, as it occurs in this affection independent of any lung disease, and also in tuberculosis of the lung during infancy.

MORTALITY AND PROGNOSIS.

Wyss gives the following table of the mortality in catarrhal pneumonia determined by various authors:

	Number of Cases.	Number of Deaths.	Remarks.
After Valleix	128	127	
" Trousseau	22	22	Measles pneumonia.
" Bouchut	55	33	
" Bartels	67	29	" "
" Ziemssen	98	36	
" Barrier	61	48	
" Steffen	66	35	
" Stiebel	16	8	
" Pfeilsticker	32	9	

This obvious difference in the report of individual authors is chiefly to be explained from the circumstance that the varieties of catarrhal pneumonia described by me have not been strictly differentiated, and that deglutition pneumonia and the pneumonia following inhalation of poisonous gases, which are usually nothing but deglutition pneumonia,—because the gases lead to insensibility, and this allows the secretions of the mouth to flow into the air-passages,—have all been included. Catarrhal pneumonia in cachectic children has an enormous mortality; that following measles, whooping-cough, and influenza is far more favorable, but the mortality varies in individual epidemics; primary catarrhal pneumonia runs the most favorable course.

No matter, however, which form of catarrhal pneumonia exists, age is always of the greatest importance in prognosis. The mortality is greater the younger the patients. According to Bartels' observation, there died of catarrhal pneumonia following measles of:

6 patients in the first years of life	6
36 patients between the first and fifth years of life	14
24 patients between the fifth and tenth years of life	9

The reports of v. Ziemssen are more favorable. His cases of pneumonia following measles were distributed as follows:

In the first year of life	6.	3 fatal cases.	3 cured.
“ “ second year of life	10.	3 “ “	7 “
“ “ third year of life	6.	1 “ case.	5 “

The cases of bronchopneumonia secondary to bronchitis and chronic catarrh were distributed as follows:

First year	14.	7 fatal cases.	7 cured.
Second year	12.	3 “ “	9 “
Third year	3.	2 “ “	1 “

The cases of bronchopneumonia following whooping-cough were distributed as follows:

First year	2.	1 fatal case.	1 cured.
Second year	7.	4 “ cases.	3 “
Third year	7.	4 “ “	3 “

The prognosis, as the preceding tables have shown, depends, first, upon the character of the disease. Catarrhal pneumonia in a previously healthy child, even when the father has been affected by syphilis, is comparatively favorable. In the cases following measles or whooping-cough several symptoms are of value in prognosis. High fever, markedly increased respiration, cyanosis, coma, make the outcome of the affection exceedingly questionable and unfavorable, especially if the inflammation has affected the greater part of both lungs. Nevertheless with all the previously quoted symptoms in previously healthy children recovery may take place. Catarrhal pneumonia of cachectic, scrofulous, rachitic children gives a very unfavorable prognosis. In the hospital, where these little patients are usually sent in a well-advanced condition of their affection, they die with the rarest exceptions. In such cases, in contrast to the previously mentioned types, temperature, respiration, and cyanosis are prognostically of no value. The temperature is but slightly increased, the respiration scarcely quickened. Pallor, emaciation, feebleness of the digestive powers, are the symptoms which, if they persist for any length of time, produce death.

DIAGNOSIS.

The diagnosis from capillary bronchitis, in the absence of physical signs which show an infiltration, depends upon the temperature. Neither a gradual rise of the temperature to 39° C. (102.2° F.) nor to 40° C. (104° F.) proves in a given case the development of pneumonia, but the subsequent course, the duration of the febrile condition, is of great importance. If the temperature for more than twenty-four hours, or even more than forty-eight hours, varies between 39° and 40° C. (102.2° and 104° F.) and over, and other conditions that may give rise to a rise in temperature, as an acute exanthem, angina, otitis media, etc., can be excluded, it is in favor of pneumonia and against bronchitis (Wyss, p. 772).

Of especial importance and not always easy is the differentiation from croupous pneumonia. An acute onset of the affection, perhaps with convulsions, in previously healthy children, very rapid rise of the temperature with slight morning remissions, absence in the beginning of catarrhal râles in the bronchi, with the presence of cough, are in favor of a rapidly beginning consolidation over one or more lobes, and of a croupous pneumonia. If the apex is affected first, croupous pneumonia is all the more certain, because catarrhal pneumonia very rarely shows itself in the apex primarily. I myself have never seen the onset of a catarrhal pneumonia in an apex.

The diagnosis becomes more difficult if the affected child comes under treatment in the course of pneumonia, and one or both lower lobes are consolidated. Then, above all, a very exact history in reference to the duration of the affection and in reference to former affections or contagious diseases, or previous catarrh of the upper air-passages, must be inquired into, which in themselves are already symptoms of a beginning catarrhal pneumonia. Should the history be entirely negative, a decision can only be reached in the further course of the affection, with close observation. Croupous pneumonia, even when it terminates by lysis, is of shorter duration than catarrhal pneumonia. In the former case dulness disappears more rapidly, provided no pleural effusion be present. In such conditions the decision upon the basis of objective phenomena is impossible, and remains impossible, as do all other conditions which were previously mentioned under History.

The diagnosis of tuberculosis becomes simple from the fact that a catarrhal pneumonia only rarely develops into tuberculosis. Positive differentiation is, however, not possible in all cases. It is impossible to diagnosticate anything but catarrhal pneumonia in a child who after cough has lasted for some time presents dulness and crepitant râles over one of the lower lobes, and yet in a case observed by me which terminated fatally the autopsy showed that there was decided swelling and caseation of the bronchial lymph-glands, and a dissemination of nearly the entire right apex with miliary tuberculosis, whereas the remaining parts of the lungs and the other organs were free from tubercles. Perhaps in the future the examination of the blood by a simple method, and in a manner not requiring too much time, will determine the possibility of showing tubercle bacilli. Nevertheless, in all such cases a decided help in differentiation depends upon the locality of the seat of the two affections. Without limiting one's self too much, it may be said: Pulmonary tuberculosis, even in children, affects the apices. Catarrhal pneumonia in children affects the bases. If there are dulness and râles present over one or both apices, the bases may also show signs of the presence of disseminated foci, and it will be correct to make the diagnosis "tuberculosis." The fact that the auscultatory phenomena, especially the occurrence of râles over the apices, are the most prominent phenomena must always be borne in mind. An empty percussion note over the apex, as has already been mentioned, may occur without the apex necessarily being affected.

PROPHYLAXIS AND TREATMENT.

As regards the occurrence of catarrhal pneumonia after measles or whooping-cough, the **prophylaxis** depends, in the first place, upon the prevention of these contagious diseases; so, then, in the existence of these affections the most favorable hygienic condition should be secured to prevent the disease from gaining access to the finer bronchi. The most important requisite for this is a sunny, well-ventilated room.

In scrofulous and rachitic children, analogous conditions should be fulfilled to prevent the occurrence of catarrhal pneumonia. Besides this, however, especial attention should be paid to the nutrition of the child. This, however, is hopeless for the majority of cases.

The **treatment** is unfortunately symptomatic, unless one is inclined, in order to fulfil causal indications, as in the case of croupous pneumonia, to employ quinin. I myself have used this drug, and in children with fever have given small doses of the neutral tannate of quinin. Children up to one year of age have received doses twice daily of 1 dcg. ($1\frac{1}{2}$ grains); older ones, 2 dcg. (3 grains). The method of using the drug has been mentioned on page 539. To lose time in discussing venesection is unnecessary, after v. Ziemssen has so conclusively proved the deleterious effects of such a treatment. On the other hand, I have, like Bartels, and after him v. Ziemssen, v. Jürgensen, and Wyss, a word to say in reference to the use of cold water.

According to the method advised by Bartels, which is based on the treatment of catarrhal pneumonia following measles, a linen cloth or a folded napkin is wrung out of very cold water and wrapped around the sick child from the back, enveloping the entire thorax. Enveloped in this way the child is laid in its bed, and in half an hour the linen, which has in the mean time become warmed, is changed for a fresh, cold one. With the use of these compresses, according to Bartels, the inspiratory movements become deeper, sleep, which sometimes is absent for several days, is restored, the temperature lowered, and the frequency of the pulse and respiration markedly diminished. Usually this condition is reached in from eight to twelve hours; sometimes, however, it is necessary after twelve to sixteen hours to repeat the manœuver, if the fever and dyspnea again become marked. In some few cases this treatment has to be repeated for several days and nights; and if the consolidation is at first very extensive, for several weeks. In all cases the patient should be carefully watched, as in this treatment the heat abstraction may be so great as to bring about collapse.

I have used this method of treatment in my little patients in the presence of marked fever, although I have not seen as prompt results as Bartels. The compress should be changed at short intervals, not only because with every application of the cold cloth a deeper inspiration occurs, and a greater amount of heat is taken from the body by frequently changing the compresses, but also because at every application the body is brought from the dorsal into the lateral position, or is raised, and in this way the disease is prevented from gaining the lowest parts of the lungs in which the circulation of the blood is most difficult. Frequent change to the erect or lateral position tends to prevent stasis of the blood. The increased pain in a felon when the arm is allowed to hang shows the significance of any interference with the return flow of blood; a stoppage in the blood-current occurring in the lowest portion of the lungs must interfere with and delay the process of recovery.

Perhaps the difference between my results and those reported by Bartels is due to the fact that I order a piece of woolen material or flannel placed around the wet application, whereas Bartels does not mention anything of this character, so that I must assume that this covering, which prevents evaporation, and therefore permanent cooling, was purposely omitted by him. But this is attended with some inconveniences; in cases with high temperature of more than 39.5° C. (103.1° F.) in the axilla, in which a lasting reduction in temperature is desirable, a general wet pack should be employed. Upon the bed-mattress a thick woolen blanket is spread; then a bed-sheet is dipped in cold water, wrung out, and placed upon the blanket and the naked child placed upon the sheet; the latter is then wrapped around the child's body so that only the head remains uncovered, and the woolen blanket placed around the sheet. It is not without value, in order to prevent a too decided reduction of the temperature, to cover the whole with a blanket so that only the head remains free. After the child has been for one hour in this pack, the wet coverings are removed and the child is thoroughly dried. Almost invariably a decided reduction of temperature is attained with this procedure; but the temperature soon rises again, so that this manœuver must be repeated twice and sometimes three times daily. The repetition of the procedure in an individual case depends upon the strength of the child, and on its comfort or discomfort during the applications.

Baths of a temperature of from 20° to 24° R. (77° to 86° F.) and of a duration of from twenty to twenty-five minutes, followed by affusions of from 10 to 20 liters of water from a moderate height, are recommended by v. Jürgensen in the case of high temperature, or if the fever is obstinate. I have not made use of the method. Only in individual cases in which there was dyspnea, cyanosis, stupor with high temperature, have I employed a bath at a temperature of from 25° to 26° R. (88.2° to 90.5° F.) five to ten minutes in duration followed by affusions of cold water upon the neck and back from a moderate height.

A direct action upon the mucous membranes of the larger and smaller bronchi is attained by the use of emetics and expectorants, on the one hand, or by means of sedatives and narcotics, on the other.

The use of emetics is not in accord with our present views. It is well to restrict their use to those rare cases in which the affection sets in very acutely and in which inside of twenty-four to thirty-six hours marked dyspnea and cyanosis are noted. The treatment is of course applicable only to previously healthy children, and must be avoided in cases in which debility is due to some previous illness. In these cases powders containing 1 cg. ($\frac{1}{6}$ grain) of the tartrate of antimony and potassium with 3 dcg. ($4\frac{1}{2}$ grains) of the powdered roots of ipecacuanha, one powder every ten or fifteen minutes until emesis occurs, are useful. I decidedly advise against a later repetition of this dose, also against the use of other emetics. I must especially warn against the employment of apomorphin, as this drug readily produces collapse.

Expectorants are frequently of use in the course of the affection. Of most value is a weak infusion of ipecac (1 to 2 dcg.—$1\frac{1}{2}$ to 3 grains —in 100 water and 20 syr. simpl., one teaspoonful every two hours). According to the age of the child, I add 1 to 3 gm. (15 to 45 grains) aq. amyg. am. It must, however, be noted that some children manifest an idiosyncrasy against ipecac. With or without vomiting a condition of exhaustion may occur, which must be referred to the action of the ipecac, as it disappears when the drug is discontinued.

As the most suitable sedative for the irritation of the mucous membrane of the bronchial tubes which shows itself in short severe paroxysms of coughing, I employ steam. The apparatus described by me in my reports in reference to the therapeutics of inhalation,[9] made by Moritz Schön in Crimmitzschau, has many advantages over basins, in which the water must be boiled. The steam may be brought into very close proximity to the patient's mouth.

Narcotics are occasionally absolutely necessary. When the child is coughing day and night, and passive hyperemia of the larynx is produced which leads to hoarseness and sleeplessness and exhaustion, I employ minute doses of morphin. Of a solution of 2 cg. ($\frac{1}{3}$ grain) morphin in 10 gm. (f℥iiss) distilled water, children two years of age receive 5 drops. This small quantity is generally quite satisfactory, but as a rule only after the first dose. The succeeding doses, which according to indications must be given twice or three times daily, must amount to 10 to 15 drops. It therefore depends upon the action of the first dose whether the succeeding doses must be increased, and to what amount.

A modification of the treatment just described is necessary in the treatment of cachectic catarrhal pneumonia, such as occurs in the course of scrofula and rachitis, as well after long-continued catarrh of the bowel. The temperature in these cases is not high, as a rule. The applications to the chest need only be changed every two hours. To the neutral tannate of quinin I add 3 dcg. (4½ grains) of saccharated iron oxid. An especially nourishing diet suitable to the infant organism is to be advised. Wine must nearly always be employed. Provided the season and the weather be suitable, children well wrapped up may remain in the open air, in the garden or in the woods, for hours in their baby-coaches.

If the catarrhal pneumonia follows influenza and runs its course with severe symptoms, I strongly advise the injections of quinin described on page 540.

If after the disappearance of the fever, in a case of catarrhal pneumonia, independent of any local conditions dulness remains, careful diet (milk-cure) and residence in a sanatorium are of value.

ATYPICAL PULMONARY INFLAMMATIONS.

The province of genuine croupous pneumonia until lately included inflammatory affections of the lung, which, in their mode of origin, their course and progress, decidedly differ from this affection. Only by very few authors who have had an opportunity of noting such conditions has the necessity of a strict division of these varieties from croupous pneumonia been emphasized. The failure to make this distinction has given rise to many misunderstandings, especially as regards the etiologic conception of croupous pneumonia itself. Because cases of these pulmonary inflammations about to be described occur in large numbers, both in individual families and in institutions, especially in prisons, making it but natural to assume that the spread is due to direct transmission, and that the disease in question is an infectious disease not inferior to erysipelas and measles in its power of transmission, the conclusion was erroneously arrived at that genuine croupous pneumonia was a disease which was directly transmissible from individual to individual. It may, however, be asserted that there is no observation on record by which this could be substantiated. In the enormous frequency of croupous pneumonia this negative statement has decided value. I have, in all, had the opportunity of observing more than 2000 cases of croupous pneumonia, and not once, either in private practice or in the hospital, saw a single instance in which the question of contagion was considered. With this it must be stated that the pneumonia patients in the hospital were in the same wards with other patients suffering from acute and chronic diseases.

This reason is alone sufficient to justify a distinct division of croupous pneumonia from the varieties which were formerly, and are even to-day, included in the same category. These varieties have been described by the authors who have especially studied them within the last twenty years as erysipelatous, asthenic, bilious, and infectious pneumonia. The clinical and pathologic differences are alone sufficient to demand a strict differentiation.

To enter into a description of the so-called "typhoid pneumonia" at this place is not necessary, although some of the varieties described under this name belong in the category of atypical pneumonias. The connection between enteric fever and croupous pneumonia has already been referred to (page 498).

The etiology alone of the pulmonary inflammations which have been collected under the term "typhoid pneumonia" shows that these varieties are essentially different. It may be maintained to-day that various bacteria may produce such atypical pneumonias. An exact limitation of the various forms will be possible only when the classification is carried out according to the bacterial causes. In the mean time we must be content with a general view of the clinical groups which are linked together by similar pathologic changes.

With the abandonment of the untenable conception "pneumotyphus" in the former sense of an exclusive localization of the typhoid process in the lungs, Friedreich has declared it to be absolutely necessary to separate the varieties to be described here from croupous pneumonia. He regards some special forms of acute pneumonia as the expression of an acute infectious process which differs in many well-marked respects from the ordinary so-called croupous pneumonia, which so frequently arises after exposure to cold, especially during the prevalence of sharp, cold, north and northwest winds. In the former the inflammation begins in a circumscribed area; by means of physical examination it may readily be determined how the process of hepatization advances from day to day. The fever frequently lasts ten, twelve, or fourteen days and longer, with undiminished intensity. The defervescence in the majority of cases occurs by lysis, which is several days in duration; more rarely by a rapidly oncoming crisis. These *wandering pneumonias* have a much more serious character than the usual forms; at the height of the process of hepatization, occasionally with the development of a more or less distinctly icteroid discoloration of the conjunctiva, the development of delirium, dry tongue, diarrhea, and occasionally loss of control of the sphincters and the appearance of marked collapse, they assume a malignant, pernicious character, which produces a certain similarity with severe typhoid affections. Remarkable to a high degree, and a proof of the infectious nature of these wandering pneumonias, is the regular occurrence during the first few days of demonstrable enlargement of the spleen, which occasionally extends from three to four fingers'-breadths below the margin of the ribs and is easily palpable. After the cessation of the fever the spleen, as a rule, returns to its normal size.

Under the name of "asthenic pneumonia" Leichtenstern has exhaustively described certain forms of the disease, and has thus thrown new light on this obscure question. Pathologically these forms are characterized by the peculiarity that the infiltration takes place very slowly, involving at first only a fraction of a lobe and then gradually becoming lobar; by flaccid hepatization; by rapid transition to gray hepatization, and, in some cases, by a marked tendency to purulent infiltration, abscess formation, and gangrene. Clinically, certain anomalies in the symptomatology are observed: the disease develops insidiously; the initial chill, the pain, the bloody sputum, are absent; the temperature curve is atypical; there is extreme prostration; and cerebral symptoms are marked. The causes of these pneumonias are either individual, such as previous chronic disease, extreme age, the use of alcohol, with degeneration in special organs, particularly degeneration of the heart, produced by this poison,—and accordingly these varieties are to be characterized as *secondary asthenic* forms; or the causes are independent of the individuality of the affected person, and are due to the causes that give rise to the pneumonia themselves, as the frequent occurrence of the disease in special places and at special times, occurring with equal frequency in the young and in the old, as well in weak as in strong individuals. These varieties are known as *primary asthenic* forms. In this form enlargement of the spleen and liver is very common. In addition, jaundice freqeuntly occurs. Albuminuria is never absent.

Primary asthenic pneumonia differs specifically from the simple form; it is a different disease, and depends upon infection with a different specific poison.

Among the varieties described by Friedreich and Leichtenstern, Kühn includes contagious pneumonia, which he had an opportunity of seeing in the Mohringer Penal Institution. He emphasizes that the pneumonias seen by him are to be strictly differentiated from croupous pneumonia. The frequent occurrence in a lobular form, the constant enlargement of the spleen, as well as the almost regular occurrence of nephritis, are in favor of a different special specific poison. Whether this may be referred to the unfavorable atmosphere in consequence of overfilling of the institution, or to an irritant contained in the working material (the weaving of cocoanut fibers, the manufacture of felt shoes), he leaves undetermined.

Kerschensteiner also observed an epidemic of pneumonia occurring in prison. He believes that the cause of this epidemic should be referred to an infectious soil. He believes it likely that the soil was at that time able to develop a pathogenic micro-organism which could not in this case be positively referred to a pneumonia patient. The autopsy showed croupous lobar pneumonia in the stage of red and gray hepatization with occasional beginning pus formation. More than this was not reported in 46 fatal cases. Nor was it stated how many autopsies were held.

The endemic pneumonia described by Müller must also be included in this category. In the house of a poor village watchman three of the four inmates and two relatives who visited them were affected, one after the other. The onset and course of the disease resembled an infectious disease more closely than an ordinary croupous pneumonia. Pneumonic sputum and pains in the side were present, but the dulness did not even extend over an entire lobe. All recovered.

Butry also describes a very severe village epidemic in which the disease developed in relatives who had visited the patients. Among the 20 persons affected, 12 were adults. Of these, 8 died. In the fatal cases icterus occurred "frequently in an intensive form."

From these communications it will be noted that all the authors mentioned have placed the cases observed by them in a special variety. Janssen, however, is of the opinion that primary asthenic pneumonia and croupous pneumonia are due to the same virus. He had an opportunity to treat bilious pneumonia in the infantry battalion which was garrisoned in Helder. The peculiarities of these cases consisted in a very marked and well-defined prodromal stage. In 7 of the 11 cases chill was absent; in none of them was herpes labialis present. The nervous symptoms were very intense. Jaundice, seen in 6 of the 11 cases, was never very marked. Albuminuria was absent but once, and as a rule was of a marked grade. The spleen was always enlarged. The temperature showed no regularity. The remissions were always very great and the drops in temperature which occurred were always interrupted by fresh exacerbations, which was due to the fact that the diseased process constantly spread to new areas of the lung. The infiltration of the lung developed very slowly, so that always several days passed before the dulness could be demonstrated by the methods of physical diagnosis. At the postmortem the picture presented by the diseased lung was not similar to that seen in croupous pneumonia. Upon the cut surface the well-known granular surface was missed; the section was smooth and of a uniform color.

After this description the etiologic identity of these cases with croupous pneumonia is not justified.

Whereas these descriptions prove that the disease must be separated from croupous pneumonia both clinically and pathologically, the following cases illustrate certain causes that do not apply to croupous pneumonia.

Ritter reports a house epidemic in which five members of the family, and twelve days later two persons who had been working in the house, were taken ill of pneumonia. Three cases terminated fatally. The autopsy showed in one case grayish-red lobular hepatization of the lungs; in the second, grayish-red serous croupous hepatization of the lungs. In many areas the macroscopic findings rather suggested catarrhal pneumonia, although microscopic examination showed that in this, as in both the other cases, a flabby croupous pneumonia had occurred. In the third case the condition of the exudate showed little that differed from the first. "Here, as there, there was beginning abscess formation." In many of the alveoli large balls of micrococci were present; the lymph system of the pleura in many cases was almost completely filled by them. "The pathologic finding, strictly speaking, allows only the diagnosis of croupous lobular pneumonia." The cause of this disease he refers to a contagium imported by birds brought from a distance or their cages.

Gastou describes 11 cases belonging to an epidemic of infectious pneumonia, whose origin apparently was due to the presence of parrots, which were newly imported from Buenos Ayres. The disease affected, without difference, young and old people; it was sufficient to have come in contact with one of these parrots to be affected by a severe illness, which even terminated fatally. After a careful inspection of the original focus of the epidemic which showed very bad hygienic conditions, and from the observation of cases in which the disease was transferred from the sick to the well, without the latter having any connection or coming in contact with the parrots, —the results of the microscopic examination yielded too few positive results to assist in explaining the affection,—Gastou comes to the following conclusions: (1) The epidemic which prevailed in the La Roquette quarter in Vaugirard, and in other places which were noted, is an epidemic of infectious pneumonia. (2) This epidemic is not the result of a disease transferred from parrots to human beings. (3) The severity of the disease may be probably referred to the unhygienic condition of the dwellings, which were overloaded with all kinds of pathogenic agents, as well as to the ease with which persons who came in contact with those affected were attacked (wine-dealers in two different localities); it is also partly due to the poor condition of health of the majority of those affected. (4) The many and various manifestations of the disease belong to the category of infectious influenzal pneumonia. (5) The only part which parrots could have played in this epidemic is the fact that they made it easy for the disease to spread; partly and especially because they made it possible for certain persons to meet, and partly by carrying the germs of the disease in their feathers in the same manner as a person may do in his clothes.

Malenchini also leaves it undecided whether the epidemic which occurred in the years 1894–95 in Florence, an epidemic of infectious pneumonia with a very unfavorable clinical course, was or was not due to parrots which had been imported.

Other observations and examination, however, leave no doubt that the disease which Morange called "psittakosis" may be transferred from parrots to human beings. The communications of Gilbert and Fournier evidently favor this view, and their reports must be communicated at this place.

On the 30th of December, 1895, Mr. K. bought three parrots. One of these he sent to a relative, Mrs. Z. This parrot died in the first days of January, 1896, and some days later Mrs. Z. and her companion were taken ill. The illness lasted several weeks. Both patients recovered; the convalescence, however, lasted a long time. Of the remaining two parrots, one died on the 3d of January and the other on the 10th, after they had appeared sad, with drooping feathers, refusing nourishment, and suffering from diarrhea.

Mr. K., the owner of these two parrots, was taken ill on the 15th of January. There was decided chilliness; upon the 20th of January the temperature rose to 40.8° C. (105.4° F.), and varied between 40.5° and 41° C. (104.9° and 105.8° F.). On the 22d of January a pneumonic area in the right base was demonstrable. Constipation, vomiting, delirium, and an appreciable quantity of albumin in the urine were the special symptoms present. Death occurred in coma, on the 26th of January.

In the case of Mrs. K., the disease began abruptly on the 22d of January. The temperature varied during the entire duration of the affection between 29° and 40° C. (84.2° and 104° F.). An affection of the lung soon showed itself, first in the right, then in the left base. With similar symptoms as in her husband's case, death occurred upon the 1st of February.

The son of these people, twenty-two years old, also was taken ill. The affection, however, was slight. Recovery soon occurred.

The two persons who died had busied themselves a great deal with the parrots—more than the others. They fed the birds, which were already sick, letting them eat from their mouths, bathed them in order to combat the diarrhea, warmed them under their clothes, upon the naked chest, etc. A further cause for the severity of the affection was the age of those affected, which was also observed in earlier observations.

As regards other varieties of pneumonia which are communicable from animal to man, it must be mentioned that Wiedenmann noted two cases of pneumonia which he observed in children and which, on the strength of the anatomic findings, he regarded as identical with tuberculosis of cattle. Positive grounds for connecting the disease with the taking of milk from a cow affected by tuberculosis could not etiologically be obtained.

The pneumonia which arises in pyemia must also be included. I have twice in pyemia, which had arisen in an acute endocarditis, and which gave rise to the formation of pyemic areas in the kidneys, been able to demonstrate at the autopsy lobar inflammation, in both cases localized in the lower lobes.

PATHOLOGIC ANATOMY.

Atypical pneumonias are lobar. Very frequently an entire lobe of the lung is uniformly affected. It is heavy, airless, and, upon section, of a dark red appearance. The structure, however, in the main, appears to be soft, similar to a diseased lung of croupous pneumonia in the stage of engorgement. In other cases there are found in the uniformly dark red tissue, which is of the appearance just described, individual areas which are more grayish, of the size of a hazelnut and larger. The boundaries are irregular and the cut surface is occasionally granular. At other times the connective tissue that accompanies the bronchi and larger vessels is greatly increased for some distance; it is then interspersed in the pulmonary tissue in the form of gray bands. Very likely a specific affection underlies this process. In the rare cases of this kind it was not possible for me to find the cause.

The gray occasionally granular areas previously mentioned have frequently led to the supposition that in this process there was more of a catarrhal pneumonia than of an affection which had an analogy with croupous pneumonia. This supposition, however, is not tenable if the condition of the mucous membrane of the smallest bronchi, and of the bronchial vessels contained in their walls, is taken as a guide. A description of these changes was given in the section devoted to the pathology of catarrhal pneumonia (p. 551); they are illustrated in figures 6 and 7, Plate 5.

In atypical pneumonia the lumina of the smallest bronchi are open. The smallest vessels and their capillaries appear almost empty in a microscopic preparation. From this the conclusion may be drawn that the tissue of the bronchial wall is not affected by the inflammation; the elasticity of the vessels has remained intact. Their contents, as well as those of the capillaries, could be expressed into the veins antemortem.

The period when these gray areas arise in the uniformly affected tissue must be taken into consideration in atypical pneumonia. For it shows that they cannot be compared to the peribronchial areas of catarrhal pneumonia. Here the appearance of these areas is the primary process; the pulmonary tissue lying between frequently remains entirely intact, or only gradually and secondarily takes part in the inflammation. In atypical pneumonia, however, the pulmonary tissue of one or more lobes is uniformly diseased, and only in the further course of the disease do changes occur in special areas, or in the form of isolated foci.

The essential difference between this variety of lobar affection and the lobar affection in croupous pneumonia consists in the absence of fibrin in the alveolar space during the entire course of the disease. The onset of the processes in many cases is exactly alike. The alveolar epithelium here also first swells and afterward coalesces, frequently into large, multinuclear cells. To this is added, earlier and more frequently than in croupous pneumonia, enlargement and thickening of the alveolar walls. This is followed in many places, but without uniformity, by an outpouring of blood into the alveoli. Among such alveoli, which are completely filled with blood, there are others, which are only filled with large alveolar epithelium and white blood-corpuscles. These differences in the individual divisions of the affected lobe explain the irregular appearance of the cut surface. Where a larger group of alveoli is almost exclusively filled with white blood-corpuscles, areas of variable extent show themselves to the naked eye, which have a gray color, standing out somewhat above the surface, and occasionally even having a granular appearance. These structural differences in the individual divisions of the previously *in toto* affected pulmonary lobe, which at first are affected as a whole, may now be looked upon as the special criterion of atypical pneumonia, and make it possible to embrace in a provisional group a number of diseases which undoubtedly in the future will be differentiated, each according to its bacteriologic cause.

THE ETIOLOGY OF ATYPICAL PNEUMONIA.

So far the bacterial findings in cases of atypical pneumoina have not been sufficiently definite, and no attempt has been made to separate the different varieties of bacteria found, so that it is as yet impossible to classify the groups of pneumonia just described. Finkler found various bacteria in the forms of the disease which he describes as cellular pneumonia and which, in the main, embrace the forms here described as atypical pneumonias, besides some others that do not, in my opinion, belong in the same category—the cellular pneumonia following measles and phlegmonous pneumonia in tuberculosis. The micro-organisms found by Finkler are:

Diplococcus, in 2 cases (not identical with Fraenkel's);
Bacillus pneumoniæ, in 1 case;
Staphylococcus, in 12 cases;
Streptococcus, in 27 cases (8 times in pure culture).

But these findings fail to explain the etiology of the individual groups, the classification of which is based on clinical observations. It is to be expected that in the future a special bacteriologic cause will be found for each variety. For the present we have the following groups:

Asthenic and bilious pneumonia;
Prison pneumonia;

Pneumonia occurring in epidemic form in special localities and directly transmissible;

Pneumonias communicable from birds to man;

Pneumonias communicable from other animals to man;

Pneumonias occurring in pyemia.

Up to the present, however, the bacteriologic investigation with special regard to this classification has been productive of but slight results.

In a fatal case of asthenic pneumonia I have myself, in microtome sections of the affected pulmonary parts stained with fuchsin, seen bacilli in the tissue which closely resembled Bacterium coli; a certain proof by cultivation was, however, not possible. Müller, in a case of pneumonia which he, it is true, regards as belonging to croupous pneumonia, found in the sputum, in the blood, and in the urine a bacillus which produced sulphuretted hydrogen. He supposes that the organism in question was Bacterium coli or one of its congeners.

Kreibich has arrived at a better result in reference to the causal significance of Bacterium coli in lobar pneumonia. In a case of pneumonia of the left base, which occurred in connection with insufficiency of the mitral valve, dilatation of the stomach, hypertrophy of the prostate, and dilatation of the bladder, in cover-glass preparations he found long staphylococci, which were arranged in small groups, 4 to 6 bacilli lying together, and was able to demonstrate them in pure cultures, taken from the lung as well as from the spinal marrow, as Bacteria coli. After intratracheal and intrathoracic injection of this bacillus into rabbits, pneumonia occurred. He concludes from this that Bacterium coli commune is capable of producing lobar pneumonia in man. As regards the mode of infection, he believes that aspiration is not impossible. In most cases of pneumonia due to Bacterium coli, there is probably a hematogenous infection, derived from the bowel or from some inflammatory process of the urinogenital tract.

As regards psittakosis,—the pneumonia which is communicable from parrots to man,—we have certain results. Nocard, in the year 1893, demonstrated a bacillus from the spinal marrow of a diseased parrot the characteristics of which he describes as follows: "It is a short, comparatively thick bacillus, with rounded ends, of extraordinary motility. It is aerobic and anaerobic, develops quickly upon most of the ordinary culture-media, provided their reaction is neutral or slightly alkaline; is decolorized by Gram's method; does not liquefy gelatin; does not produce lactose fermentation, and does not coagulate milk." Gilbert and Fournier obtained from the dealer from whom the three parrots referred to on page 584 were bought another bird, which had died after a few days of severe diarrhea. All the abdominal organs were markedly hyperemic. The peritoneum showed isolated ecchymoses; the spleen was swollen; the bowel was hyperemic to a marked degree, and ulcerated in places. In the blood, in the spleen, in the spinal marrow, and in the bowel they found a bacillus which was identical with the one described by Nocard. In the patients treated by them (see p. 584) they failed to find this bacillus either in the sputum or in the blood derived from the finger by the prick of a pin. From the former only virulent pneumococci and streptococci could be isolated. In two patients of Matthieu's also,—man and wife,—that suffered from psittakosis, they could not find the bacillus in the blood derived from a vein, or in the sputum, or in a small pleural effusion of the woman, or in the blood derived by puncture from the spleen, three days before death. But they were able to isolate it from the blood of the heart of the woman, which was obtained at the autopsy, and to prove its complete identity with Nocard's bacillus. The virulence for various animals (parrots, mice, rabbits, etc.) was alike severe. As regards the other peculiarities of this bacillus, they determined that no indol reaction occurs in a peptone solution, and in culture upon potatoes it resembles the Escherich bacillus. It contains from 10 to 12 spores, similar to Eberth's bacillus. They are, however, more friable and are easily loosened. Besides this, they determined the effects of serum diagnosis, in which they used the serum of well persons and of typhoid patients. They added to 20 drops of a twenty-four-hour-old bouillon culture one drop of serum, which was taken from an enteric fever patient, and saw a very plain clumping of the bacilli occur; this, however, was less voluminous and plain than under similar conditions with the typhoid bacillus, and the bacilli did not lose their motility. On adding the serum of a healthy person, they do not appear to show any modification.

Upon the basis of these results the authors arrive at the conclusion that the disease in question is due to a bacterium which can be positively differentiated, which is frequent in parrots, and easily communicable to man.

[It is plainly seen that the term atypical pneumonia will not be employed when we can recognize the micro-organism which causes the infection and attribute to it a symptomatology which would correspond to the specific infection, just as we have a symptomatology for a pneumococcus infection. All inflammations of the lungs will be called infections, and we will have a pneumococcus, a streptococcus, a staphylococcus, etc., infection.—Ed.]

SYMPTOMATOLOGY.

Every individual variety of atypical pneumonia presents peculiarities in the duration and severity of the disease, according to the individuality and the age of the one affected. To this there is added the dissimilarity of the course depending on a dissimilarity in the causes of the disease. From this a variety of symptoms result, which make it exceedingly difficult to give a comprehensive description.

The fever may occur acutely, with a chill, just as in croupous pneumonia. The temperature then rises rapidly to a marked height. Except in abortive varieties, in which, after from twenty-four to forty-eight hours, the temperature becomes normal, the fever may be continuous, lasting several days, and—provided death does not occur at the acme of the disease—may terminate either by lysis or by crisis. "Every severe case does not, however, terminate with the first defervescence; in case of relapse, the fever, after the first critical fall, again completes a pneumonic cycle; or, if concomitant pleural or pericardial processes become prominent, the temperature curve is quite atypical" (Kühn).

In other cases the disease begins with all the signs of general malaise and a gradual rise in the temperature, so that only temperatures of 40° C. (104° F.) and over are not reached before the fourth day. In such cases the objective changes in the lungs are not prominent; two or three days may go by before a certain proof of consolidation is possible.

Very early, in many cases, the mucous membrane of the pharynx shows itself affected. In the patients observed by Kühn, angina and catarrhal stomatitis formed an integral part of the picture of the disease, and were only rarely missed. This affection was usually noticeable from one to two days before the onset of the fever, and in some few cases led to a high grade of swelling and diphtheritic deposits. The stomatitis in one year (1876) regularly went on to stomacace, or a mild form of scorbutic gingivitis. Gastou remarks that in a few instances the disease assumed a different form, in that after a severe chill, which was accompanied by headache and pains in the neck, an angina occurred with a yellowish, apparently gangrenous coating which was almost adherent, resembling large aphthæ, but was not accompanied by swelling of the lymphatic glands of the neck. Simultaneously with this angina, congestion of the lungs occurred, accompanied with marked debility and a sensation of great anxiety.

Examination of the lungs makes it possible to determine objective changes in every case. It is true that there may be a lapse of two to three days from the onset of the disease before the objective changes can be determined. Then, however, there are always signs of pulmonary consolidation present. Only these are in the rarest cases as characteristic as in croupous pneumonia. This is due to the anatomic condition of the diseased pulmonary parts, which deviate markedly from those which occur in croupous pneumonia. There are differences in the results of auscultation and percussion between a hepatized pulmonary lobe in croupous pneumonia and a splenified lobe in atypical pneumonia. At least these designations, which have been in use for a long time, appear to me to be most characteristic for the clinical condition describing the differences in these diseases.

Percussion rarely shows well-developed dulness; an empty note, with or without a tympanitic admixture, frequently occurs. Just so auscultation in only a few cases shows loud bronchial breathing; as a rule, there is but soft bronchial respiration and crepitation. The sputa are rarely characteristic. Rusty sputum only rarely occurs.

The spleen is almost always enlarged to a greater or less extent.

Catarrh of the bowel occasionally occurs. The patients of Gastou all had profuse diarrheal stools of yellow appearance.

The kidneys are frequently affected. The urine contains more or less albumin. At the autopsy parenchymatous nephritis is found (Kühn).

Upon the skin patches of roseola show themselves, occasionally also petechiæ (Ritter, Gastou).

The nervous system is frequently affected. The gravity and severity of the affection are shown in, and on the other hand are partly responsible for, the profound involvement of the nervous system. As a rule, stupor and delirium occur early. Ritter noticed in one case jactitation; later, severe delirium and attempts at flight.

In the seven cases in which Kühn was able at the autopsy to investigate the condition of the brain, four cases showed an exudative meningitis, more or less well developed; once, in the case of a patient who during the typical fever period showed severe cerebral symptoms and later succumbed to an extensive empyema, there was found thickening of the pia as well as marked cloudy swelling in the surrounding vessels; twice there was decided hyperemia of the meninges, serous engorgement, also hyperemia of the brain and a slight lessening of the ventricular fluid.

Of the other complications the most frequent is pleurisy of either a purulent or a serous nature. Pericarditis is rare.

PROGNOSIS AND DIAGNOSIS.

The reports of all authors agree that the disease, no matter which form of atypical pneumonia described it may assume, is very severe, and the mortality is very great. Early involvement of the nervous system, with marked symptoms, large quantities of albumin in the urine above all, may render the course exceedingly unfavorable.

In the *diagnosis*, main attention must be paid to the differences between this disease and croupous pneumonia. In practice it will frequently be very difficult to diagnosticate the first case correctly. If, however, a number of cases occur in the same institution, in a single family, or in persons who have directly or indirectly come in contact with the affected person, there is every reason to believe, according to recent experience, that an atypical and not a croupous pneumonia exists; and the local conditions must therefore be thoroughly searched for any clue to the etiology.

The objective symptoms of the disease itself must be carefully utilized in the diagnosis. Apart from the results of auscultation and percussion, as well as the relatively early development of severe cerebral symptoms, and of albumin in the urine, I should like to lay special stress on the necessity of examining the sputum in reference to its bacterial contents. It is very easy to prepare cover-glass preparations from the sputum, to dry them over a flame, then stain them for five minutes with a solution of fuchsin-rubin (0.01 in 100.0 aqua destillata), rinse in water, dry, and mount in Canada balsam—a method which is equally useful for pneumonia diplococci and for influenza bacilli.

In croupous pneumonia there is found, with the simplest caution (the possible avoidance and mixing of the pneumonic sputum with the mucus of the mouth), especially if rusty sputum is available, at least an overwhelming number of the characteristic Diplococcus pneumoniæ. In atypical pneumonia, in all likelihood, other bacteria will be found in the majority. Such a result of course in itself proves nothing definite in regard to an etiologic connection between these bacteria and the disease. It, however, justifies the supposition that the affection is not croupous pneumonia, and gives occasion to carefully weigh all pathogenetic circumstances to arrive at a correct diagnosis. Positive proofs I cannot certainly bring, because I have seen but few cases of atypical pneumonia, which belonged partly in the province of asthenic pneumonia and partly in the province of pyemic pneumonia. The fact that in very intricate cases of pneumonia I was able to make the diagnosis between influenza pneumonia and croupous pneumonia only by means of examination of the sputum, leads me to give the advice always to make a bacteriologic examination of the sputum.

The diagnosis from enteric fever has but slight actual interest, since we know that the localization of the typhoid poison in the lungs in the sense of a primary process must be looked upon as unproved (compare above, p. 498). It is not possible in such cases—that is, in disease of the bowel in which a pneumonia occurs—to demonstrate the presence of typhoid bacilli; and without such proof the cases observed by Rodman of infectious pneumonia cannot be looked upon as typhoid. Nor can Kühn bring sufficient proofs for the conversion of contagious forms of pneumonia into typhoid or typhus fever; even if among 12 cases that terminated fatally "in the typical fever period," he 8 times saw the solitary follicles, as well as Peyer's patches, swollen, prominent, pigmented, but not ulcerated.

PROPHYLAXIS AND TREATMENT.

The etiologic conditions described allow many useful prophylactic measures. If in a closed institution a case of atypical pneumonia is diagnosticated, naturally strict isolation of the patient is necessary. Even if several inmates of the institution are attacked before the disease is correctly diagnosticated, much trouble may be averted by making use of this measure. Besides, measures are to be taken to improve the sanitary conditions of the house. A similar procedure is necessary when the disease originates in a single family or in a single locality. Only those having to do with the nursing and observation of the patient should be allowed access to him.

Further, it must be noted whether or not an epizootic disease exists in the dwelling or in the workshop of the patient. Up to the present, the communicability of the disease-producing agent from animals to man has been definitely determined only in the case of parrots. Gilbert and Fournier advise strict supervision of the importation of parrots; the prohibition of the sale of parrots, especially sick parrots, by itinerant venders; rules for bird merchants and possessors of parrots in reference to isolation of sick animals; and instruction of the public in reference to the danger of the communicability of the disease from parrots to man.

As regards treatment, it coincides with that of croupous pneumonia. In the want of better therapeutic measures, in severe cases the subcutaneous use of quinin (p. 539), with stimulants, should be strongly advised.

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HYPOSTATIC PNEUMONIA.

"The engorgement of blood or serum, often found in the posterior parts of the lungs of cadavers, has heretofore been considered a consequence of the gravitation of fluids occurring after death, or at least during the last moments of life. This was formerly my opinion also, but later observation convinced me that hypostatic pneumonia precedes death for quite a time, and in many cases is the consequence of the fatal issue. It has been frequently mistaken for bronchitis, with which it is likely to be complicated."

It was with these words that Piorry for the first time transferred the condition we are about to discuss from the sphere of postmortem, or at least agonal, findings to that of clinical medicine. "Only the obscurity of the symptoms could give rise to the supposition that hypostatic congestion of the lungs was an agonal phenomenon, the physical signs proving that this condition is present even before life is extinct. The stethoscope and pleximeter give excellent proof of this; the former, by demonstrating absence or decrease of the respiratory sounds, the latter by aiding us to discover a constantly increasing dull note; at the same time there is noted an increase in resistance, becoming daily more manifest. In those areas in which dulness upon percussion is noted engorgement will be found at the autopsy."

But Piorry also asks the question: "Is hypostatic congestion of the lungs inflammatory in character?" and answers: "This pathologic condition will almost always be found subject to the laws of gravity. In such cases the heart is flabby and may be easily torn by the finger. The disease appears to be caused by weakness and to be due to the influence of gravity, but it may be complicated by an acute inflammation. For after the hypostatic pneumonia has lasted some time, one will find the lungs to be in a state of red, gray, or straw-colored hepatization similar to the condition occurring in acute pneumonia. The symptoms observed during life also justify the same conclusions. For a short time after the first signs of hypostatic congestion the heartbeats are increased, the pulse is harder, the respirations are more frequent, the skin is congested, and the face is reddened. Sometimes the sputum becomes rusty; in other cases pleurodynia is noted. In this manner the pneumonia, originally not inflammatory, may assume the character of an acute inflammation of the lungs."

These views of Piorry, according to my opinion, are still indisputable, as will be substantiated by the following discussion.

PATHOLOGIC ANATOMY.

The affection begins at the lowest part of the lungs and advances upward as well as forward, following the laws of gravity; it does not conform to the lobar construction of the lungs. Its almost exclusive limitation to the lower lobe is due to the fact that the vertical dimensions of this lobe are about equal to the antero-posterior dimensions measured from the lowest points.

The affected parts are almost entirely airless, and upon handling feel somewhat tough and doughy. Such a condition is termed *splenization.* According to Rindfleisch, this splenization appears to offer extremely favorable conditions for the occurrence of inflammatory changes. It may take the place of the initial hyperemia of the acute inflammations, and may be succeeded by lobular or lobar, but always catarrhal, infiltrations. According to his statement, this is the case in the hypostatic pneumonias of enteric fever and marasmus.

The further anatomic changes in the course of this affection, as they have been observed in aged individuals, have been depicted to perfection by Hourmann and Déchambre. While according to their statement there are no essential differences from croupous pneumonia during the first stage, during the second the inflamed lungs are neither so heavy nor so enlarged as when equally affected areas are involved in ordinary pneumonia. The affected parts do not sink entirely in water, although they sink to a greater depth than do normal lungs. Two varieties of this stage are noted in the aged. The first is characterized by the perfectly smooth appearance of the lungs when incised. The cut surface is homogeneous, and from it oozes a bright red, tenacious fluid which is not frothy. These impermeable areas are sometimes elastic, sometimes entirely soft. The second form shows the cut surface granular, but the granules are considerably larger than in hepatized lungs of younger individuals; they are at times regularly circular and of equal size; at other times exceedingly varied in shape and size, according to the manner in which the senile state has made itself manifest in the structure of the remaining parenchyma. Very rarely are the hepatized parts softened to the same extent as in ordinary pneumonia, but they are always more moist.

The third stage of inflammation is a repetition of the two forms just mentioned. Sometimes the cut surface is even. Irregular, narrow, striped spots of a puriform character can be noted, which, by moderate pressure, may be displaced up to the cut surface. At other times the surface shows the same more or less coarse granules seen in the second stage. The softening in this case is at least as considerable as in ordinary pneumonia.

This description should be correct for all other cases of hypostatic pneumonia, even should it occur in younger individuals in connection with acute or chronic diseases. Of course, the above-described changes, owing to the shorter duration of the affection, develop comparatively rarely. Under these circumstances the lung does not show more than a congestion at the onset of the condition, giving the dependent parts a darker aspect and causing a decrease of air in the alveoli. On the cut surfaces the blood oozes in single drops from the smaller vessels, and this is followed by the further change, characterizing—to use Piorry's expression—the transformation of the hypostasis into hypostatic pneumonia: "The air contents disappear entirely, the tissue is heavier, pressure of the finger perforates the cut surface, and no more blood appears. The latter is caused by the walls of the smaller vessels having lost their elasticity under the influence of the inflammation."

The bronchial mucous membrane of the diseased parts is affected always in the form of a severe hyperemia, from which a purulent catarrh may develop. The above-mentioned narrow-striped spots of puriform character probably correspond with the contents of finer bronchioles.

The participation of the pleura is generally insignificant. Frequently slight ecchymoses appear on the pleuræ, and sometimes fresh fibrinous deposits. Hourmann and Déchambre, in 60 cases of old people examined, found pleuritic pseudomembranes in 38.

Pleural effusions which, according to several authors, sometimes appear in connection with hypostatic pneumonia, are rare. I have never observed such a case. Piorry says: "The frequency of pleurisy in acute and real pneumonia, on the one hand, and its rarity in hypostatic pneumonia on the other, is certainly one of the most important distinctions between these affections."

Microscopic examination will still more explicitly illustrate the character of hypostatic pneumonia and its distinction from other forms. According to the method mentioned in the foot-note on page 396, I have made this examination, with the following results:

The most important change appearing in all cases refers to the alveolar epithelia. The latter invariably contain brownish, friable masses, but the form and the size of the cell protoplasm are not sufficiently preserved to correspond with a normal cell. The friable material is also found outside of the epithelia and free in the alveoli.

This change differs but little from that of the first stage of croupous pneumonia; in the latter the epithelia are unchanged in form and size and do not degenerate, whereas in hypostatic pneumonia the nuclei of the alveolar epithelia undergo changes that gradually destroy the cells. When stained with Biondi-Heidenhain's stain, they become brown, and not blue, like the nuclei of normal epithelia; moreover, it is impossible further to stain the nuclei with the same stain. The alveoli contain only non-nucleated flakes as large as alveolar epithelia, which fact I was able to demonstrate clearly in a case of hypostatic pneumonia complicating ascending paralysis. Although I was unable to observe an absolute disintegration of the nuclei into granules as a preparatory stage of nuclear disintegration, nevertheless, I have no doubt that the entire process is identical with the changes*[10] observed in kidney cells after ligation of the kidney. This latter process I have designated *coagulative necrosis*, in the sense that Weigert uses the term.

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*The small figures throughout the text are references to the Bibliography on page 592.

This change of the epithelia, never absent in any case of hypostatic pneumonia, is sometimes accompanied by a complete congestion of the alveolar capillaries which in normal lungs cannot be demonstrated by the above-mentioned method of staining. It differs from the congestion of the capillaries in croupous pneumonia in so far as in this affection the capillaries are constantly very much wider.

In rare cases the alveoli may also contain fibrin, the condition differing from croupous pneumonia only by the fact that the fibrin-plugs are less abundant. A disintegration of the alveolar walls may also take place, causing the fibrin-plugs of several alveoli to coalesce.

While all these changes are uniformly distributed over the entire diseased portion of the lung, in accordance with the duration of the illness or the rapidity of the process (?),—that is to say, while a necrobiotic process at first exclusively affects the alveolar epithelium, and later, if the pathologic process in the epithelium progresses more slowly, hyperemia of the alveolar capillaries and a transudation of blood-corpuscles and fibrin may be added,—later on only single groups of alveoli appear filled with round cells. In connection with the above is the fact which has already been emphasized by Förster ([15], page 247), that the cut surface in hypostatic pneumonia is never as coarse and as granular as in ordinary pneumonia.

I must leave it undecided whether extensive lobar infiltrations may follow as Rindfleisch asserts; but I can deny the occurrence of catarrhal infiltrations which the same author assumes. In no instance was I able to demonstrate the essential condition of catarrhal pneumonia on which I laid stress in my communication on infantile pneumonia—namely, the inflammatory hyperemia increasing to hemorrhage of the capillary terminations of the arteries in the walls of the finest bronchial twigs.

ETIOLOGY AND PATHOGENESIS.

Experience teaches that hypostatic pneumonia occurs: (1) In old individuals, spontaneously or, more frequently, when they are forced to remain in bed for weeks, as, for instance, on account of fracture of the neck of the femur. (2) In the course of protracted infectious diseases; for instance, enteric fever. (3) According to my observations, it develops with striking frequency and rapidity—*i. e.*, within a few days—in the train of severe cerebral and spinal affections. I have seen hypostatic pneumonia appear during the first two or three weeks of ascending paralysis, of hemorrhages into the medullary pia mater, and of a fracture of a cervical vertebra; moreover, I was able to demonstrate it by the autopsy. I have also observed hypostatic pneumonia to follow other cerebral diseases; for instance, apoplexy, in which it is comparatively frequent.

It should be admissible to regard not only a weakness of the myocardium, as has been correctly done so far, but also a weakness of the vascular system in general, as the essential condition for the origin of hypostatic pneumonia. Therefore, one should look also into the local condition of the vessels for the cause of the detrimental influence exercised by the gravitation of the blood upon the nutrition of the lowest parts of the lungs. This local condition depends upon a decrease of the elasticity of the blood-vessels, which, under normal conditions, is a considerable factor in bringing about the flow of the blood from the arteries into the capillaries and beyond them into the venous system. Atheromatous changes in the vessels in senility should especially be taken into consideration in this connection.

According to Piorry, embarrassment of the circulation in the dependent parts of the lungs may be observed even in healthy individuals. He states that in all individuals, after the nocturnal dorsal decubitus, the percussion note and the respiratory sounds are decreased in the morning, principally upon the right side, even in the strongest person. But an absolute confirmation of this statement by other authors is still lacking. Jürgensen gives a more convincing example as proof of the importance of gravitation in the origin of hypostasis and its consequences. The leg of a typhoid patient with thrombosis of the crural vein was put into a box and the patient brought into the left lateral position. The ensuing hypostasis occupied a narrow strip in the lateral section of the lung which corresponded exactly with the most dependent part of the organ.

If, according to Piorry, "hypostatic pneumonia" attacks principally the right side, it "seems to be caused by the weight of the liver, which is not counterbalanced by the weight of the heart and spleen. The heaviest part of the liver is on the right side, and as this part forms the end of a lever represented by this organ, it must draw the body over to its side."

The pathogenesis of the affection may be traced to an insufficient function of the vessels, or, more correctly, of the vascular walls which causes congestion, with a detrimental effect, principally on the lowest parts of the lungs. The consequence of this congestion is deficient nutrition of the pulmonary tissue, principally of the alveolar epithelia. In these the disturbed nutrition will cause a necrobiotic process. If this take place very rapidly, a destruction of the epithelia will follow, principally by means of coagulation necrosis (disappearing of the nucleus probably preceded by its disintegration). If the progress be a little slower, it is possible that an inflammatory irritation may be set up in the vessels by the epithelia, followed by exudation of blood-corpuscles and of fibrin, in smaller areas also by the emigration of white blood-corpuscles.

Therefore the process of hypostatic pneumonia should not be looked upon as inflammatory at the onset. It is only when, before the nutritive disturbance in the alveolar epithelium has advanced too far, the cells of the latter irritate the alveolar wall, especially the alveolar capillaries, that inflammatory changes will set in (see page

392) which conform with those occurring in croupous pneumonia, but are neither as intense nor as extensive.

SYMPTOMATOLOGY.

As hypostatic pneumonia in most cases is a complication of severe affections not necessarily of the lungs, and as at the onset it does not show any noteworthy subjective symptoms, it is likely to escape observation without an objective examination. "Even in the aged who do not suffer from any other disease but their debility, and dislike of exercise, very often neither difficult respiration nor cough nor sputum is observed, although the engorgement of the posterior pulmonary lobes may have reached a high degree" (Piorry).

As a remarkable initial symptom I have observed a high degree of exhaustion, out of all proportion to the previous course of the respective basal disease, which could not very well be explained, either by the disease itself or by any peculiarity of the fever connected with it. Daily observation of the patient may sometimes enable us to determine such a difference in the general state of health and leads us to make an examination of the most dependent pulmonary parts.

It will certainly be safer always and conscientiously to examine the lungs in all exhausting affections and exhausted patients, and not only in the aged. At the same time, the erect position in bed is of some therapeutic value.

Only in rare cases a slight increase of the already existing fever or a slight rise of temperature in an apyretic patient betrays the onset of hypostatic pneumonia.

The objective findings alone are decisive. Without exception there exists a more or less well-marked dulness at the base of a lung, which generally ascends as high as the lower third of the scapula. According to my experience, the right lung is more frequently, or at least in a higher degree, affected than the left.

In a diagnostic sense it is of special importance to remember that the dulness slightly decreases in intensity toward the lower edge of the lung, and acquires a slightly tympanitic quality. The presence of this sign greatly facilitates the differentiation from pleural effusions and hydrothorax, without which it may not always be definitively determined.

The dulness is accompanied by diminished respiratory sounds and weakening of the pectoral fremitus, rarely with light bronchial breathing. If, at the same time, fine crepitation occur, it depends upon the catarrh that exists in the smaller bronchi. If this is not present, or if the disease has complicated another affection which so far has not yet affected the lungs, it may be possible that cough is absent, during the entire course, even in cases in which a *restitutio ad integrum* occurs.

Sputum need not be expectorated at all, and it is rarely ever of a hemorrhagic character. I do not think it is quite justifiable to assume a causal connection between the dyspnea and cyanosis which sometimes develop and hypostatic pneumonia. These symptoms are more readily explained by the basal disease. The weakness of the vascular system which gave rise to hypostatic pneumonia is in itself liable to produce such manifestations.

The prognosis depends entirely upon the causal disease. The severity of the affection and its influence upon the action of the heart and the vascular system—in the aged this influence alone—determine the degree of the structural changes in the lung, and in connection herewith the possibility of complete recovery.

PROPHYLAXIS AND TREATMENT.

The principal object should be to limit the severity and duration of any disease that is capable of giving rise to hypostatic pneumonia.

If an old man, on account of a fracture of the neck of the femur, has to remain in bed for weeks, it is to be feared that hypostatic pneumonia may set in, especially if extensive atheroma of the vascular system exists. As in this case a change of posture in bed is not possible, a shortening of the stay in bed, with application of proper walking apparatus, will be of the greatest advantage.

If in an acute, long-lasting disease—as enteric fever—hypostatic pneumonia threaten, it is of importance to counteract as much as possible the injurious influence of the toxin of fever as well as the effect of the fever itself. Tepid baths with mild cold douches, which induce deep inspiration, are in place here. The patient should also be induced to change his posture in bed frequently.

Whenever possible, Jürgensen's advice should be followed and energetic action of the respiratory muscles should be brought about. He causes patients who are able to do so to take methodic, deep inspirations with the aid of the auxiliary muscles of respiration. The patient sits erect, takes hold of a support with the hands, and breathes a certain number of times in a minute.

In cases of cardiac asthenia, stimulants such as brandy or champagne are to be given. Not infrequently the use of digitalis is indicated. It may always be prescribed when the pulse is frequent and small, it being immaterial whether it is irregular or not. No other remedy will better stimulate the circulation of the blood and thus prevent congestion—an essential condition for the occurrence of hypostatic pneumonia; and if congestion has already caused hypostasis, no other remedy will better remove it. Even then, when hypostatic pneumonia has developed, the use of digitalis may be able to influence the issue favorably, provided the underlying original disease will admit of such a possibility.

ASPIRATION PNEUMONIA.

A few remarks will have to precede the description of this disease. In the first place, regarding the name. It is frequently called "deglutition pneumonia," but the name is not comprehensive enough, because it embraces only a small part of the causal conditions. It is true that in laryngoparalysis, which may either be local or caused by an affection of either the vagus nerve or of its branches, liquids during the act of deglutition are bound to enter the trachea, and from there find their way into the alveoli. The morbid changes thus produced in the pulmonary tissue are, of course, brought about by deglutition, which means that the fluids have taken the wrong course. But it is possible that identical changes in the pulmonary tissue can take place without any process of swallowing, solely by the process of inspiration. If a man fall into the water and remain under water for some time, he inspires only water instead of air. In a state of unconsciousness the deglutition movements may stop entirely. The saliva secreted in the mouth is changed into a frothy fluid by the inspired and expired air, and more or less of this fluid will be inspired into the pulmonary alveoli through the trachea. The last-named occurrences are the principal causes of the disease.

For this reason, and also not less because an entering of the fluids through the paralyzed glottis and their advancing into the alveoli is only possible by means of the respiration, it is advisable to drop entirely the name deglutition pneumonia and substitute "aspiration pneumonia," as is so frequently done at present.

Then, of course, the aspiration of arge solid bodies that remain fast in thê bronchi has to be distinguished from the aspiration of liquids containing bacteria or microscopic elements which may succeed in entering the alveoli. Only the latter is to be considered here.

But a precise definition is also required in another respect. Hitherto aspiration pneumonia has generally been erroneously identified with catarrhal pneumonia, and in text-books on medicine as well as on pathologic anatomy the two affections have been treated in common. The only reason for identifying them is found in the identity of the gross anatomic changes. In doing so the heterogeneous original causes, and in connection therewith the differences as regards age in the affected individuals (catarrhal pneumonia mostly affects children), as well as the dissimilarity in the course and termination of the two diseases, were completely overlooked.

Nor is elucidation of the question brought about even by a study of the changes in the pulmonary tissue after section of the vagus. According to Traube's generally accepted interpretation, these changes are caused by fluid from the mouth entering the finest bronchial twigs and the alveoli; for, according to Frey's investigations, vagus pneumonia is closely related to catarrhal pneumonia of infants as described by

Colberg. After bilateral section of the vagus he found processes in the lungs which were manifested by hyperemia of the parenchyma and abundant discharge of serous fluid into the bronchi, by cloudy swelling of the epithelium, by the appearance of numerous lymphoid elements in the vessels, by collapse of the hyperemic tissue of the upper lobes, by the presence of a true inflammatory infiltrate, principally of the hepatized parts, and, finally, in cases of long duration, by participation of the interstitial tissue in the inflammatory process.

I shall dwell more explicitly upon this point later on, when I describe the changes caused by the cutting of both vagi, and more precisely demonstrable with the microscope, and I shall have to prove the identity of vagus pneumonia and aspiration pneumonia as it appears in human beings as well as the differences of the two and catarrhal pneumonia.

In the first place, observations in human beings are to be considered.

Buhl, in his discourse upon pneumonia, makes a remark with reference to discriminating between deglutition pneumonia and catarrhal pneumonia. His "reflections upon the derivation of lobular foci of suppuration from a displacement of the purulent mucous secretion out of the finer bronchi into the alveoli, induce him to touch briefly upon other foreign bodies inspired into the bronchi, and their inflammatory influence." He refers principally to small bodies which are inspired into the smallest bronchi and pulmonary vesicles. In these he includes the blood that escapes from the vessels of the mucous membranes and small pieces of croupous membrane which are drawn into the pulmonary tissue. But also without hemorrhage, without manifest croupous or diphtherial affections in the upper bronchi or pharyngeal spaces, there appear in rare cases in the pulmonary tissue exactly the same areas, which differ from the lobular purulent areas of so-called catarrhal pneumonia in so far as not only the alveolar contents show purulent conditions, but also the alveolar wall and its bronchial wall are disintegrated and of a yellow pus color. The areas have something quite peculiar and characteristic about them, which is proved by the fact that they contain a nest of schizomycetes or fungi.

Furthermore, Nauwerck describes two characteristic cases of aspiration pneumonia induced by ether narcosis, which "were most likely due to an immediate aspiration of the infected material into the pulmonary parenchyma." In one case death occurred on the fifth day of the disease. The lungs contained numerous, somewhat prominent, coarse areas about as large as a hazelnut. The pleuræ showed hemorrhagic and fibrinous exudations. The second case did not terminate fatally until after two and a half weeks. Besides numerous lobular pneumonic areas, there was an abscess the size of a hazelnut on the right lower lobe which entered into the pleura and from which projected a large necrotic pulmonary lobe.

I myself had the opportunity of observing cases of aspiration pneumonia that appeared in the course of poisoning by illuminating and mine gas, and the making of carbolic acid. The investigation showed results that may help to elucidate the question.

PATHOLOGIC ANATOMY.

The macroscopic examination of the lungs of individuals that died within the first days after the onset of the disease proves the existence of small lobular areas in the dependent parts of the lungs. At first glance one might incline to the belief that these areas are the result of the same process that takes place in catarrhal pneumonia. But a closer examination with the naked eye will suffice to show some differences. The areas look more pale gray; they are less firm and coarse and their peripheral sections still contain some air in spite of their pathologic appearance.

But the process need not be limited to the dependent parts and the lower lobes. I infer this from an observation which Langerhans published and in which an extensive disease of the lungs took place after carbolic-acid poisoning. But I must state right here that Langerhans himself does not interpret this case as one of aspiration pneumonia, but rather traces the changes to the indirect influence of the carbolic acid, and regards them as a consequence of the resorption of the poison from the intestine. He substantiates his opinion by calling attention to the fact that in his observation the numerous areas were spread over every part of both lungs, while so-called deglutition pneumonia, as is well known, is mostly found in the middle, and then in the posterior and lower parts of the lungs. Against this view, furthermore, is the short time (twenty-four hours) which elapsed between the taking of the poison and the occurrence of death; also the uniform development of the areas and the absence of every characteristic symptom in the bronchi.

But it appears to me that Langerhans' principal argument against the development of aspiration pneumonia is not tenable in view of Stubenrath's experimental examinations and my own observations in human beings. Stubenrath has produced aspiration pneumonia in rabbits by immersing them in different fluids, and in one case has observed red hepatization and exudation after only five and one-half hours; in another case even death occurred after two hours in consequence of fully developed pleuropneumonia. But in these experiments diseased areas were found also in the upper lobe. Here, as well as in human beings, the posture of the body during inspiration may be not without influence. Langerhans traced the affection of the upper parts in both lungs of the first of his two cases to the fact that the patient was a pregnant woman who occupied the dorsal position; but he explains it as being due to the direct influence of the carbolic acid which ran down into the sloping bronchial branches.

The author's report of the condition of the lungs in his second case is quite typical of the macroscopic appearance. The report reads as follows:

> "The pulmonary layer of the pleura, as far as there are no adhesions, is smooth, moist, and glistening. Almost everywhere in the left lung thickening is felt; on the other hand, crepitation can still be elicited almost all over. After viewing the cut surface one can see that a great number of the lobules, principally in the middle and superior parts of the upper lobe, are hepatized centrally, and only a more or less narrow peripheral part of the lobules contains air. The hepatized areas have all a grayish-red color and a very slightly granular cut surface. Almost all the lobules in the middle and lower parts of the lower lobe are hepatized; and many hepatized lobules may also be found in the upper and anterior parts of the lower lobe. All the hepatizations are a little flabby in the center, slightly coarser than in the periphery. Over the right lung the contiguous pleura of the upper and lower lobe appears dry. The connecting pulmonary tissue is hepatized. On the cut surface centrally hepatized lobules can everywhere be seen. Exactly the same results are found on incising the anterior and posterior parts of the right lung. The hepatizations are of exactly the same character as those in the left lung; only in the middle lobe the lobular areas commence to be confluent, but here also the hepatizations are somewhat flabby."

In the further course of aspiration pneumonia small necrotic areas are formed which, according to the kind of bacteria that entered the lungs, lead either to gangrene or to abscesses. Kreibich describes a case (No. 27) in which, in connection with a compression of the larynx by carcinomatous glands, with carcinoma of the lungs, "numerous lentil-sized abscesses appeared in the left lower lobe. Upon pressure they, as well as the bronchi, discharged thick, grayish-yellow, fetid pus, and two large abscess cavities were found in the lowest parts, surrounded by consolidated tissue."

In the case described by Orth as "mycosis septica in the newborn," which may be included here, the infant lived only three and a half days, and yet a ripe abscess as large as a cherry-stone was found in the lower edge of the right lower lobe.

The finer bronchi are probably exclusively affected. They show a congested mucous membrane and may be filled with a purulent fluid. The larger bronchi are also sometimes diffusely reddened.

After some duration of the disease the pleura will be affected; more or less extensive fibrinous exudations with hemorrhages into the pleura proper, which at the onset correspond to the pulmonary foci underneath, form the transitions to pleuritic effusions that mostly become purulent. A purulent pulmonary area may break through into the pleura.

If large effusions into the pleural cavity occur comparatively seldom, this is mainly due to the rapid unfavorable termination of the disease. The microscopic examination gives exact data for the determination of the anatomic condition, especially for the distinction between the changes taking place in aspiration pneumonia and those in catarrhal pneumonia. Regarding the latter, I have furnished special anatomic criteria in this work on page 555, and elaborated them in several points in a lecture[12] before the Society of Naturalists, at Brunswick. I particularly emphasized the fact that in this process, which originates in the bronchial mucous membrane, the hyperemia of the vessels supplying the bronchial mucous membrane is to be considered inflammatory, because I have found in many cases of catarrhal pneumonia, in addition to hyperemia extending into the capillaries, hyperemia, and extensive hemorrhages in the walls of the bronchi and their surroundings. Such hemorrhages, however, can only appear in consequence of an inflammatory disease of the vascular walls.

I was further able to prove that in catarrhal pneumonia the primary process upon which the rest depends takes place in the bronchial walls, and only through them infects the surrounding tissue, the alveoli. The latter are only affected secondarily.

The inflammation spreads from without, inward.

Of all these characteristic changes of catarrhal pneumonia, no trace is found in the lungs of persons dying of aspiration pneumonia.

The microscopic examination that I made with the aid of the staining process mentioned on page 396 gave me results that unquestionably distinguish aspiration pneumonia from the catarrhal form. In the former the finer bronchi, especially their vessels, appeared to be entirely unaffected in the different diseased areas; the changes concerned principally the alveoli and the interalveolar tissue. But within this locality differences existed that require a more minute description of the individual observations.

One of my cases was a man, twenty-nine years of age, who after typhoid fever was attacked by descending pneumonia of the right side with consecutive empyema. The empyema was evacuated through resection of the ribs, but in spite of this the patient grew worse. During the last days of his life he was mostly somnolent. Upon autopsy numerous grayish-white, airless, sometimes slightly granular looking areas were found in the lower lobe of the otherwise apparently healthy left lung. The formation of these areas I could readily surmise to be due to the aspiration of secreted material from an abscess in the right lower lobe of the lung, and this supposition was confirmed later on by the results of my microscopic examinations.

In microscopically examining these areas numerous crystalline formations in the alveoli were conspicuous. They were mostly of a rectangular shape with irregular edges. An exact chemical test was not possible because only the finished microscopic preparations were left of the whole specimen. Furthermore, the alveoli appeared to be entirely filled with cellular elements which were stained red-brown and which corresponded with the swollen alveolar epithelia. In most of them nuclei were no longer visible, and when they were present, with a few exceptions they proved to be unstainable. They were of a pale appearance. Sometimes two or three such nuclei were in one cell. Here and there the protoplasm had the appearance of honeycombs. Only small quantities of fibrin were found in the alveoli. The alveolar capillaries appeared mostly conspicuously hyperemic.

Another case was that of a robust man, twenty-two years of age, who, when bathing, went into shallow water head first and struck bottom. He remained under water for some time. He was very quickly revived, but from that time on he could not move his legs, and immediately felt great weakness in his arms. The next day fever and tracheal râles set in, and seventy-two hours after the accident death occurred. Besides a fracture of the third cervical vertebra and compression of the cervical spinal cord, a large number of airless areas, varying in size from that of a pea to that of a hazelnut, were found in the lower lobes of the lungs. These areas were gray in color and in striking contrast to their very congested surroundings.

The microscopic examination showed in these areas large groups of alveoli completely filled with white blood-corpuscles, interspersed with red ones; the vessels of the interalveolar tissue were highly congested, while the alveoli in the neighborhood of these alveolar groups contained almost nothing but red blood-corpuscles. Only scanty remnants of the alveolar epithelium were visible.

Finally, I shall explicitly describe a case of aspiration pneumonia which set in after carbolic-acid poisoning. A woman, seventy-seven years of age, weary of life, had taken a large quantity of crude carbolic acid. A few hours later she became unconscious and was taken to the hospital, when tracheal râles were at once noted. Here her stomach was washed out with plenty of water until the fluid ceased to smell of carbolic acid. Subcutaneous injections of oil of camphor also were used. The sensorium became clearer, but the stertorous breathing continued, the pulse remained small, and the extremities were cool. Later on abundant stools of a brown color appeared and vomiting occurred once. With renewed disappearance of consciousness, increasing frigidity, and decreasing pulse, death occurred twenty-eight hours after the intoxication. The autopsy showed that in the entire intestinal tract, especially in the pharynx and esophagus, no sign of hyperemia or even of eschar was visible. But the mucous membranes of the laryngeal orifice and of the vocal cords were conspicuously turgid; the entire bronchial mucous membrane was highly congested. The lungs were edematous all over; the left lower lobe and the lower part of the left upper lobe were consolidated although still containing air in some places.

The microscopic examination demonstrated the changes observed in the left lung to be caused by a very large number of small compressed areas in which a central nucleus and two zones could be distinguished in every case. The former embraced from 2 to 20 alveoli which were completely filled with red blood-corpuscles. Other visible alveolar epithelia appeared to be without nuclei. The zone nearest the central nucleus also showed a congestion of the alveoli, but this was mostly caused by red blood-corpuscles with swollen alveolar epithelia between them. Only a few white corpuscles were present. The alveoli in the exterior zone contained much fibrin in reticular form which surrounded isolated swollen alveolar epithelia. Normal pulmonary tissue was found between the different compressed areas.

But in many places the central part of the area showed further advanced changes. The separate white blood-corpuscles were invisible; the entire part was coagulated into a structureless mass and stood aloof from the inner zone, at different places showing a wide fissure. Sometimes separate elastic fibers, which were torn out of their connection with the other tissue, extended into such a fissure.

In spite of the difference of the microscopic changes in the three cases described above (which correspond with other cases observed), a uniform interpretation of the entire process does not present any special difficulties.

Although in quite a number of places the finest bronchioles contain amorphous masses, probably derived from the disintegration of leucocytes, the essential part of the disease, upon which its anatomic and clinical character as well as the termination depend, ends in the pulmonary parenchyma. The alveoli are always the seat of the affection, and frequently the interalveolar tissue is also affected. Either a swelling of the alveolar epithelia with disintegration of their nuclei sets in, if the disease takes a rapid course,—that is, a complete coagulation necrosis, accompanied by intense hyperemia of the alveolar capillaries,—or, during the subsequent course, or if the duration is protracted, an exudation of white blood-corpuscles into the most affected alveoli occurs, filling them completely. But in such cases a peripheral inflamed zone appears, the alveoli of which then contain distended epithelia and numerous red, and a few white blood-corpuscles. Only little fibrin can be discerned between these cellular formations. Sometimes this zone is surrounded by another outer one, the alveoli in which also show pathologic changes in the shape of a dense net of filaments which surrounds the swollen alveolar epithelia and a few red and white blood-corpuscles.

Whenever intensity and duration permit the full development of the process, the consequence is that the central alveoli which are congested with white blood-corpuscles—in other words, the original focus of the disease—are cast off. This change may take place very rapidly, as the above-cited case of carbolic-acid poisoning proves. Within twenty-eight hours there appeared a line of demarcation where the continuity of the interalveolar tissue was interrupted, so that only the elastic fibers of the alveolar wall extended across this line.

It is evident that abscesses may later develop in these places. The necrotic nucleus, separated by a line of demarcation from the morbid but still living tissue, must necessarily lead to suppuration, eventually to gangrene, if the patient lives long enough.

That this description of the pathologic process is correct is further shown by the results of my experiments in vagotomy, performed on rabbits. If any more proof is needed to show that the inflammatory changes in the pulmonary tissue which follow bilateral vagotomy are caused by the aspiration of fluid from the mouth, I believe irrefutable evidence is furnished by the following experiments, which seemed to me necessary to explain the morbid process in diseases due to the inhalation of dust, which will be described later.

In some of the experiments I put charcoal of linden-wood, such as may be bought in any drug-store, mixed with glycerin to the consistency of an extract, into the mouth of a rabbit; in other cases I used sulphid of mercury, prepared the same as the charcoal; and immediately afterward performed bilateral vagotomy. This done, a small quantity of the glycerin compound was again put into the mouths of the rabbits, and this process was repeated twice daily as long as they remained alive. On account of the promptness with which death occurred after bilateral vagotomy of the rabbits, the charcoal or cinnabar could only be given three or four times.

In the first place, vagotomy without synchronous rubbing of pigment in the mouth of the animal resulted in pneumonia rapidly terminating in death, in one case within thirty, in the other within fifty hours. The following observations were made:

1. Large white rabbit. May 28, 1898. Bilateral vagotomy. Death after thirty hours. The left thoracic cavity contains an abundant quantity of transparent serum. The pleura is covered with a fibrinous exudate. The left upper lobe is absolutely airless, firm, and very congested. Gray condensations of pinhead-size show through the surface as well as on the cut surface. The superior half of the left base shows the same conditions. The lower half is permeated by hemorrhages, but otherwise contains air. The right upper lobe, except a small part of the lowest section, is also without air, but slightly congested; a number of gray condensations on the cut surface can also be seen. The right middle lobe is almost completely atelectatic and contains only a few small pneumonic areas. The right lower lobe in its upper part shows the same conditions; the larger lower part is fairly well filled with air.
2. Large gray rabbit. May 28, 1898. Bilateral vagotomy. Death after fifty hours. In the left thoracic cavity copious transparent serum is found. The left apex, except a small posterior part, is airless, congested, and shows a few gray areas on the cut surface. The left base is also airless except a small frontal part, with numerous small aciniform gray areas. The right lung shows in all lobes the same conditions, except the middle lobe, which presents a dark bluish-black appearance and contains on the cut surface numerous gray areas which in some places are confluent. The epiglottis and the vocal cords are pale, the laryngeal and tracheal mucous membranes are much reddened.

According to the above account, there is a conspicuous difference in the anatomic conditions between the experimental results and the conditions in human aspiration pneumonia. In the latter the lower lobes are almost exclusively the seat of the pathologic changes; in the rabbits, the upper as well as the middle lobes appear to be most affected. But this difference is only apparent and well adapted to confirm the explanation of the processes in aspiration pneumonia. If the supposition is correct that in this disease fluid from the mouth flows through the paralyzed glottis into the bronchial twigs and into the alveoli, and thus produces the disease, then the lowest parts of the lungs must be affected first and primarily. In human beings the lowest parts are the bases; in quadrupeds the apices, especially so in rabbits, which mostly crouch forward when lying down.

The locality of the affection was the same in those rabbits which received the thick glycerin compound almost simultaneously with the vagotomy. I shall only mention one experiment in which red sulphid of mercury was used:

On June 17, 1898, bilateral vagotomy was performed with a pair of dissecting forceps and a compound of glycerin and sulphid of mercury put into the mouth. The latter was repeated on June 18th at 7 and 11 A. M. Temperature 39.1° C. (102.4° F.). On June 19th at 6.30 and at 11 A. M. the same doses were applied; temperature 39.2° C. (102.6° F.). On June 20th, about fifty hours after operation, the rabbit was found dead.

The left lung is bound to the thoracic wall by slight fibrinous adhesions, and is entirely airless except a small part of the lower edge. The airless parts are pale and emphysematous; congested areas, mostly aciniform, everywhere show through the pleura. The completely atelectatic pulmonary tissue shows likewise numerous vermilion colored areas on the cut surface. Wherever this redness is not prevalent, the tissue has a hyaline gray appearance.

The apex of the right lung, except the lower emphysematous edge, is, like the middle lobe, of a dark livid appearance. On the surface and cut surface vermilion areas show in large quantities, and likewise on the tissue which also appears livid; the base contains air, is edematous, and very emphysematous. Abundant quantities of red sulphid of mercury are found in esophagus and stomach.

This experiment, as well as another one with a compound of glycerin and charcoal, proved conclusively that ample quantities of the contents of the mouth were inhaled into the lungs. In these cases it is really a question of actual deglutition pneumonia. The act of deglutition brings only a part of the contents of the mouth into the esophagus, another quite considerable part going through the paralyzed glottis into the lungs.

The microscopic examination of the changes in the pulmonary tissue produced by the experiment plainly sustains the justice of the opinion that an absolute conformity exists between the pulmonary changes caused by bilateral vagotomy and those seen in human aspiration pneumonia, the origin of the latter being immaterial. The process in the lungs of the rabbits was also mainly caused by changes in the alveoli; here also groups of alveoli, or single ones, were filled with granular cells; their surroundings showing analogous though lesser changes. These latter consisted mostly in a considerable swelling of the alveolar epithelia and more or less copious hemorrhages into the alveoli and the surrounding tissue. Worthy of mention also is the finding of amorphous material in the finer bronchi and of fibrinous plugs in the finer vessels. In some places the blood must have coagulated in the smaller vessels before life was extinct, for they contained a uniform red mass in which no morphologic elements could be distinguished.

Catarrhal pneumonia as a consequence of bilateral vagotomy is out of the question when the definition given on pages 551 and 609 is taken into consideration. These experiments do not bring about any other changes except such as correspond in every way with those of human aspiration pneumonia, as described.

PATHOGENESIS AND ETIOLOGY.

The affection is caused by infectious material being aspirated into the alveoli through the bronchi. One of the most frequent exciting causes is the occurrence of tracheal râles, the result of an exceedingly plentiful, mostly acute, secretion of the bronchial mucous membrane with loss of consciousness. The secretion of the bronchi enters the oral cavity through the larynx, mixes with the saliva, and is partly drawn back again into the trachea. In the same manner the small corpuscular elements present in the mouth,—for instance, the epithelia of the mouth,—as well as the bacteria from there, find access to the lungs.

Until recent years ether narcosis, which apparently, and with good reason, we have entirely discarded in Germany, represented a typical process of this kind. After Poppert and Mikulicz had connected the occurrence of bronchopneumonia with this method of anesthetization, and after the former found that existing statistics did not prove the alleged superiority of ether over chloroform, Nauwerck, as mentioned, described explicitly two fatal cases following ether anesthesia. He correctly ascribes the origin of the pneumonia to autoinfection from the oral cavity. The narcosis removes the barrier between the infected oral cavity and the bronchi, and the secretion of both, moved up and down by respiration, becomes mixed with infected material and gets into the pulmonary parenchyma.

According to my own observations the process in ether narcosis takes place exactly as reported by Nauwerck. When present at operations, I have had repeated opportunities to observe the tracheal râle during the narcosis. The bronchial secretion is bound to be forced up into the oral cavity during expiration, and to be drawn down again during inspiration, thus causing a highly infectious fluid to be taken up by the pulmonary parenchyma. The possibility of severe injury to the pulmonary tissue is therefore evident and cannot be guarded against. Healthy lungs, on the one hand, short duration of the narcosis on the other, are probably the only conditions to prevent dangerous consequences.

Lindermann has furthermore proved by experiments that during ether narcosis a part of the contents of the mouth enter the bronchi. He put carmin powder into the mouth of a rabbit, and anesthetized it for an hour with ether. The animal died with intense râles during the etherization, and carmin powder was found both in the lumen of the trachea and in the larger bronchi. [Whitney considers ether pneumonia a microbic disease, and preventable by antisepsis of mouth. Anders points out that the infection is not derived from the inhaling apparatus, but that causal influences are season (in 80% of his cases ether pneumonia occurred at the time of the year when pneumonia is most prevalent), the depression of a prolonged operation, as of the abdomen, and previous disease of the nose, mouth, and bronchial tubes.—Ed.]

Bruns, it is true, ascribes the occurrence of severe bronchitis or lethal bronchopneumonia to the use of ether of a poor quality; by inhaling pure ether only a speedily passing irritation of the mucous membrane is produced. He hopes to overcome the bad secondary effects by using a good preparation exclusively. I doubt whether his hope will be realized, because I have observed an analogy between the symptoms in ether narcosis and the symptoms manifested in the respiratory organs after carbolic-acid, illuminating-gas, and sewer-gas intoxications. This analogy is so conspicuous that any specific influence of the ether may be excluded and in every case the aspiration of infected material from the oral cavity must be regarded as the cause of the disease.

The observation and demonstration of the above-mentioned heterogeneous cases of intoxication make Grossmann's explanation also inadmissible. He says: "Concentrated ether fumes cause a reflex spasmodic stenosis of the larynx, and simultaneously an increased swelling and secretion of the mucous membrane. Furthermore, they cause, by direct influence on the velum palati, base of the tongue, and epiglottis, anesthesia or paralysis of these parts."

Tracheal râles as well as loss of consciousness were present in the cases of intoxication observed by me (twice from swallowing of large quantities of concentrated crude carbolic acid, once from illuminating gas, once from a fall into a sewer) just as in ether narcosis. The unconsciousness is sufficient to abolish the reflex action of the nerves supplying the glottis, and consequently to bring about aspiration of fluid from the mouth.

I have also seen diffuse congestion of the bronchial mucous membrane in carbolic-acid intoxication, exactly as has been observed after ether narcosis. Is, therefore, not the uniform explanation admissible, for the latter cases as well as for carbolic-acid intoxication, that the material aspired from the cavity of the mouth causes an irritation of the bronchi which in the larger ones leads to catarrh, and in the finer ones to considerable exudation?

With reference to pathogenesis, next to the entrance of oral secretion into the bronchi, during inspiration, occurrences have to be noted in which the inhalation of air is impossible and nothing but watery fluid is introduced into the bronchi by the involuntary act of respiration. An instructive instance of this theory is the case, mentioned above, of a man who, having suffered a fracture of the cervical vertebra while diving, remained under water for some time, and who, in consequence, was attacked later by aspiration pneumonia. The cases observed in the newborn and described as septic pneumonia belong to this category. Taking former observations into consideration, Silbermann reports four cases of his own. Referring to the observation of Orth, who ascribes the origin of the disease to the transmission of bacteria from the blood of the mother to that of the child, and assuming that the bacteria reach the lungs only through the blood, Silbermann concludes that septic pneumonia of the newborn is caused by the aspiration of putrefied genital secretions (or by the inspiration of air infected by the mother's septic disease). Sometimes it is complicated by pleural affections, rarely by other organic diseases; in contradistinction to other septic processes in the newborn which are characterized by the appearance of multiple foci. According to Silbermann's statements, the alveoli and bronchi of children who have died of septic pneumonia are filled with masses of bacteria. [Reference can be made at this point to Steele's valuable paper on pleuritis in the newborn. The twenty-five cases he collected had in nearly every instance catarrhal pneumonia, and he agrees with Orth in supporting the claims of Fischl that the pneumonia and the pleuritis were the result of a general infection by pyogenic micro-organisms. The mode of infection was through the umbilicus, or during intra-uterine life, or through infected breast-milk, and in four cases out of twenty-five by aspiration.—Ed.]

Local affections of the bronchi, complicated by ulceration, are another cause of aspiration pneumonia; so, also, is the breaking through of affected areas from the surroundings of the bronchi into these, and the infection of one lung, furnishing secretions which are drawn into the other previously healthy lung.

Kreibich gives characteristic examples of aspiration pneumonia, valuable on account of the bacteriologic examination as well as of the pulmonary changes following rupture of the diseased areas into the bronchi. In his eighth and twelfth cases it was carcinoma which caused perforation; in his eleventh, a diverticulum of the esophagus had the same effect.

Henoch's opinion regarding the influence of diphtheria upon the occurrence of aspiration pneumonia deserves special mention in a pathogenetic respect. He correctly ascribes the putrid bronchitis as well as the circumscribed gangrene of the lung, occurring in small areas in such cases, to the aspiration of diphtherial or gangrenous particles of tissue which not only set up an inflammation, but also directly infect the finer bronchi and the surrounding parenchyma.

Not less remarkable is Bäumler's communication on the inflammatory changes which took place in the course of tuberculosis and which he considers an acute inhalation or aspiration inflammation, both because of the onset of the process and of the lobular and vesicular localization of the affection. It is also possible that in patients in whom the pulmonary tuberculosis was absolutely latent, and in whom, either in one or in both apices, old cavities surrounded by callous tissue were found, purulent or hemorrhagic contents of these cavities may have been aspirated. The direct occasions of carrying the bacterial inflammatory cause which may exist, in addition to Bacillus tuberculosis, are hemorrhages from tuberculous sections and the aspiration into the finest bronchial twigs of blood containing the morbid matter.

In all cases the co-operation of the bacteria is necessary to produce inflammatory disease of the lungs. Upon their kind and properties depend the form, intensity, and severity of the disease. Some influence must also be attributed to the complicating affection that is the cause of the process of aspiration, as, for instance, in the case mentioned above, of fracture of the cervical vertebra in consequence of a dive into the water.

The bacteria enter the respiratory tract mostly with the aspirated fluid or with the aspirated corpuscular elements. An observation of Hecker teaches that such microscopically small bodies may advance as far as the alveoli. Five minutes after death of the mother by eclampsia, a child was delivered by Cesarian section and made to breathe by the proper measures. But it died after thirty-four hours. A dark turbid exudate was found in the left pleural cavity, covering the base of the lung, which was stiff, airless, and in a state of red hepatization. "The microscope did not show any pus corpuscles, but numerous large squamous cells, which could originate only in the mouth and the pharynx of the child."

But if the occurrence of aspiration pneumonia in the newborn be comparatively rare, this is principally due to the fact that during labor pathogenic bacteria but rarely enter the respiratory tract in spite of the frequent aspiration of fluids. This view is sustained by the fact that most children who develop aspiration pneumonia were born of septic mothers. According to an analysis of Silbermann, including twelve cases, nine, possibly ten, mothers suffered from severe septic infections. Most frequently a mixture of different kinds of bacteria is involved, and the share in the affection of the single species is difficult to demonstrate. Kreibich found, for instance, in one of the above-mentioned cases of perforating carcinoma: (1) Micrococcus pneumoniæ (numerous foci); (2) Bacterium coli commune; (3) a sarcina, and (4) (in sections) Micrococcus tetragenus. A particularly rare occurrence might be the finding of Bacillus subtilis, which I located in the repeatedly mentioned case of aspiration pneumonia after diving. Nothing but the characteristic Bacillus subtilis with spores was found in smear preparations of the affected lungs. There is but little doubt that the bacilli entered the respiratory tract with aspirated water, because this bacillus exists in water. Gireaudeau and Renou examined water from two different sources of which patients had drunk who were affected by cholera nostras. One kind contained staphylococci, the other Bacillus subtilis. It is true that my findings do not prove that this species of bacillus is pathogenic. Even if the inspired fluids are free from pathogenic bacteria, infectious processes may take place—namely, when the aspirated fluid acts as a chemical or mechanical irritant to the mucous membrane of the respiratory tract or the alveolar epithelia, and reduces their normal power of resistance against the influence of pathogenic bacteria, because, as is well known, normal respiratory tracts also contain bacteria.

Von Besser examined, with all precautions, the bronchial secretions of ten men shortly after death, and found Diplococcus pneumoniæ three times. The cases were fracture of the cervical vertebra, tuberculosis of the peritoneum, and enteric fever. Dürk confirmed these results by extensive examinations. Not only did he find different bacteria in human lungs, which macroscopic as well as microscopic examinations demonstrated to be free of any inflammatory deposit, but he also observed pathogenic bacteria in the lungs of cattle, sometimes in considerable numbers. He adds: "It seems evident from these results that the lungs are not the germless organs which they are generally supposed to be, but, on the contrary, pathogenic germs are very frequently found on the inner pulmonary surface, which evidently get there with the air-current."

To be sure, it must be mentioned that, according to Hildebrand's statement, the microbes contained in the air we breathe do not reach the deeper air-passages, but under normal circumstances are nearly always retained in the nasopharyngeal space. From his examinations Klipstein came to the conclusion that the trachea, the bronchi, and the lungs of healthy animals (rabbits, cats, dogs) under normal circumstances are free from germs. But these observations, even though they do not invalidate the results of the experiments mentioned, which were carried out with all necessary precautions, do not disprove the connection between the aspiration of oral secretion and the spontaneous entrance of bacteria into the air-passages, on which the present argument is based; much less so, since Klipstein by his experiments has also found that bacterial inhabitants of the cavities of mouth and nose may reach parts of the respiratory apparatus to which, under normal circumstances, they have no access, after these parts have become diseased.

· Would it not be more correct to assume that bacteria may enter the bronchi under all circumstances, but will only find favorable conditions for multiplication if an affection exists, since in health they are expelled by the respiratory organs, or neutralized in some way, as, for instance, by absorption?

[It will be observed that aspiration pneumonia can be employed only to suggest the mode of entrance of infective agencies or of materials giving rise to trauma whereby infection is invited. Hence, as with catarrhal pneumonia, the realization that the process is infectious and that a correct nomenclature can only be based upon the nature of the invading micro-organism—a pneumococcus, a streptococcus infection, etc., for instance—would remove the confusion that now exists. —Ed.]

SYMPTOMATOLOGY.

The symptoms take different forms according as the disease appears in persons up till then healthy, or occurs as a complication in other affections which existed previously. If a carcinomatous disease of the esophagus cause a perforation into the trachea, and a mixture of fluids and solid particles is inspired into the lungs, grave consequences must result. But these do not always manifest themselves by a special change of the subjective condition. Cough, with expectoration, and dyspnea may have existed before the perforation occurred. Only a regularly conducted objective examination of the lungs will lead to the demonstration of the changes which agree with those caused by primarily occurring aspiration pneumonia, and which will be described later on.

Whenever the disease occurs in healthy individuals from the aspiration of fluid, no matter whether this fluid comes from the outside, as after a fall into the water, and in the newborn intrapartum; or consists of a frothy mixture of saliva and mucus from the trachea aspirated during unconsciousness,—for instance, in ether narcosis, in poisoning by illuminating gas or carbolic acid,—the most conspicuous symptoms are those which indicate an abnormal quantity of fluid in the larger bronchi. Moist crepitant râles are heard over the lungs, both anteriorly and posteriorly, because the physical conditions greatly favor a propagation of the sounds in all directions on account of the firm, resonant bronchial walls.

If the causal conditions are borne in mind, such objective signs will lead to the conclusion that fluids have entered the lungs, although further changes may not follow. Especially in people who fall into water and who have aspirated some of it the râles may disappear within a few hours or within a day, and absolute health may follow.

In other cases local changes may take place in the pulmonary tissue. Over one or both lower lobes a dulness on percussion exists, but it does not extend over the entire lower lobe; the respiratory sounds have decreased; catarrhal sounds are audible. Consequently it is already a question of an inflammatory change of the pulmonary tissue setting in. But even in this stage a *restitutio ad integrum* may occur, and fever need not necessarily prevail.

A speedy recovery depends, in the first place, upon the quantity of fluid inspired into the lungs; and, secondly, upon the bacteria that have entered the lungs with the fluid. Undoubtedly the species of the bacteria is of the greatest importance for the course and the termination of the affection.

The best proof of this theory is found in Silbermann's statistics of the above-mentioned twelve cases of aspiration (septic) pneumonia in the newborn. Of the 12 mothers, 10 were septic; 6 of these died. If we consider that the possibility of aspiration intrapartum is the same in all newborn infants, whether the mothers pass through a normal puerperium or suffer from puerperal affections, the overwhelming occurrence of aspiration pneumonia in children born of septic mothers can only be explained by the fact that in these cases bacteria of a very virulent kind have been inspired with the fluid, while in other cases the fluid does not contain any pathogenic bacteria or only such as are of a less virulent character.

In the majority of cases in which the above-mentioned objective changes of the pulmonary tissue are manifest, principally if fever sets in directly, no quick regression takes place; the symptoms of inflammatory processes last longer in these cases.

The consecutive order of the symptoms so far mentioned—*i. e.*, the early crepitant râles over the larger bronchial twigs and the following pneumonic dulness—may be well illustrated by a carefully observed case of Silbermann of a newborn child whose mother died of puerperal peritonitis on the third day postpartum.

The child, examined immediately after birth, did not show any disease except râles over the trachea and the large bronchi. During the evening fever set in (38.4° C.—101.0° F.) and the child refused food. Coarse bubbling râles were now heard over the trachea, without dyspnea. On the next morning the fever had increased and severe cyanosis of the face and dyspnea set in. A careful examination of the child showed coarse bubbling râles over the trachea, medium-sized and fine moist râles in the fossa supraspinata and infraspinata on both sides, but no decided dulness. Pulse 120, cardiac sounds loud and clear; mucous membranes of mouth and pharynx congested and swollen, but no other organic affections. The stump of the umbilical cord showed nothing pathologic. A little urine was discharged, showing heavy sediments and containing small quantities of albumin. The dyspnea occurred in both respiratory phases equally, respiration was performed by means of the auxiliary respiratory muscles. On the evening of the second day the temperature had reached 40.2° C. (104.4° F.), with considerable dyspnea, respiration 60, pulse 160. Fine râles were heard in the lower dependent parts of the lungs, and posteriorly on the right, near the fossa infraspinata, dulness could be observed. The child died on the following morning with symptoms of most marked dyspnea. Autopsy showed the lungs poorly retracted, emphysematous at the upper edges, of a red brown color, very plethoric, and in the right middle and lower lobe, as well as in the left base, of considerable consolidation. A frothy blood-colored fluid oozed from the cut surface. The mucous membranes of the trachea and bronchi were very congested and swollen, and covered with a tough glairy mucus far into the finest twigs. The mucous membrane of the mouth and the pharynx was likewise much congested and swollen and showed numerous epithelial defects. The pleura pulmonalis was much injected and covered with small ecchymoses.

In this case only râles over the trachea and large bronchi were observed on the first day of life. Not until the evening of the second day were fine râles heard over the dependent parts of the lungs and dulness elicited over the right fossa infraspinata. Consequently, on the first day the symptoms of fluid in the bronchi were present, and not until the second day did the symptoms show the inflammation caused by this fluid.

The following case, observed by myself, will best characterize the course of aspiration pneumonia in adults.

On March 4, 1881, Charles S., tool-smith, and his wife were found in their residence, apparently lifeless. The man was unconscious, stertorous; the woman lifeless, in postmortem rigidity; a blackish fluid had oozed from the mouth of both. A vessel containing partly burned coal was found in the room. A note they had left stated that the couple had decided to commit suicide.

After an attempt had been made to revive the man and some milk infused into his mouth, he was taken to the hospital at noon. He gave the impression of utter debility and was unable to move. A brownish-black mass had dried on his cheek and underlip which must have come from his mouth. The pupils were narrow and rigid. His face was pale, slightly cyanotic, and assumed a bright red color after a cold douche in a tepid bath. When questioned, the patient was now able to tell his name in a very low, hardly audible voice. He was able to swallow if a fluid was carefully put into his mouth. His urine contained no albumin. Temperature in the evening, 37.9° C. (100.2° F.).

March 5th: Light now caused a reaction of the pupils, which were a little wider. Patient could not move his lower extremities. Their sensibility was reduced. He was almost motionless. Respiration shallow and frequent; he talked indistinctly; took a little of the fluid offered. Pulse 90, regular, a little stronger than the day before. Dulness right base posterior; slow bronchial respiration and râles. Moderate cough, no expectoration. Temperature 38.4° C. (100.1° F.) in the morning, 38.7° C. (101.7° F.) in the evening.

March 6th: An extended necrotic discoloration appeared over the sacrum. Paralysis of the legs continued. Speech plainer and stronger. He did not believe his wife was dead, and denied any attempt at suicide. Temperature 40° C. (104° F.) in the morning, 39.2° C. (102.6° F.) in the evening.

March 7th: Dulness in right base posterior advanced up to the spine of the scapula. Over this section moderately loud bronchial respiration was heard, and also ringing râles. No expectorations. Stool after castor oil. Temperature in the morning 38° C. (100.4° F.), in the evening 38.7° C. (101.7° F.).

March 8th: Great weakness. Pulse worse. Fœtor ex ore. Incontinence of feces and urine. Temperature A. M., 38.8° C. (101.8° F.); P. M., 39° C. (102.2° F.).

March 9th: The fetor of the exhaled air could be noticed at a distance from the patient. No expectoration. Cyanosis and somnolence set in. Urine contained some albumin for the first time.

Temperature A. M., 38.4° C. (101.1° F.); P. M., 39.1° C. (102.4° F.).

Tracheal râles set in during the following night and death occurred on the morning of March 10th in coma. Autopsy on March 11th, twenty-six hours after death. Robust body in postmortem rigidity without edema. Diffuse livid discoloration existed in the dependent parts, several lentil-sized suggillations were found between the scapulæ; over the sacrum were necrotic areas the size of the hand. The skin over the heels showed bullæ formation; the anterior surface of both thighs had an appearance like goose-flesh; the skin over the abdomen showed green discoloration. The subcutaneous fat was well preserved, the muscular structure strong, dry, light red.

The pericardium was not covered by the lungs. It contained 60 c.c. of clear serous fluid. The heart was of normal size. The right auricle and the right ventricle contained chicken-fat clots and reddish-blue masses of cruor.

The myocardium was dull, pale, and flabby. The intima of the aorta and valves showed ecchymoses; the latter otherwise intact. The lungs collapsed but little. The left pleural cavity contained some blood-colored fluid. The left lung was voluminous, very hyperemic and edematous, but otherwise did not show any focal disease. The right lung was also voluminous; the upper lobes contained air throughout, but were edematous. In the anterior section of the middle lobe was an area the size of a hen's egg close under the pleura which was discolored whitish-gray. This area was of blackish-green color and in a smeary condition, very fetid. A similar area as large as a man's fist was in the posterior section of the middle lobe. The latter was markedly decayed, tore when separated from the pleura over it, was slightly agglutinated to the pleura costalis by fibrinous masses, and gave rise to a cadaverous smell. The other parts of the middle lobe were entirely airless, moist on the cut surface, granular, and of a gray appearance. The lower lobe was also airless, the cut surface smooth, of a gray-greenish color. At its base were several necrotic areas, some of them as large as a cherry-stone.

The mucous membrane of the bronchi and trachea was intensely congested throughout. The posterior half of the left vocal cord showed a smeary looking ulceration which extended to the processus vocalis. The perichondrium of the latter was lacking. An ulceration of scarcely lentil size was found on the corresponding part of the right vocal cord, exposing the bare cartilage.

The spleen was 15 cm. long, 9 cm. broad, and 3 cm. thick; the capsule could be easily stripped off; the substance pale and coarse on the cut surface. The right kidney was 12 cm. long, 4.5 cm. broad, 2.5 cm. thick, and of the same appearance. The pelvis was distended to the size of a hen's egg and filled with transparent urine. The ureters did not show any changes. The bladder contained transparent urine. The middle lobe of the prostate was slightly enlarged. The stomach, much dilated by gas, had a slightly mammillated mucous membrane of pale appearance. The intestine did not show any abnormality. The liver moderately large, of coarse consistency, the acini blurred. The gall-bladder contained but little transparent, tenacious bile.

The entire clinical course in this case, as well as in those belonging to the same category, depends essentially upon the inflammatory disease of the pulmonary tissue, the location of which is dependent upon the aspirated fluid. This is mechanically bound to reach the lowest parts, and therefore one or both lower lobes are affected in every instance. However, from the position of the frequently unconscious patient, the upper lobe may also be affected. In the case just described the middle lobe was chiefly involved. It is true that in a longer duration of the disease the inflammation may advance upward, and consequently spread without reference to the course of the inspired fluid.

Without exception, a more or less pronounced dulness over one or both bases is one of the objective symptoms. Auscultation shows bronchial respiration, which, however, is rarely as loud as in croupous pneumonia, because the dulness is not as uniform as in the latter; a complete diffuse filling of the alveoli with exudation does not take place. Besides, râles are always heard over the diseased parts, because the affected finer bronchi are in a state of catarrh; they secrete mucus, which causes the auscultatory sound. The râles are medium-sized, if not coarse. [In ether pneumonia Anders shows that pain is the most common symptom, fever is usual, dyspnea is not urgent, and cough is slight or absent, while the pulse is rapid, small, and compressible. The physical signs are the most common phenomena.—Ed.]

The cough is insignificant, owing to the short duration of the disease. But if necrotic or gangrenous disintegrations of the pulmonary tissue take place, it may become very severe and the previously scanty expectoration may considerably increase. According to the effect of the different bacteria, the sputum will assume a putrid or fetid smell, which will be disseminated by the exhaled air. [In Drummond's cases (nine) the fever was irregular in course.—Ed.]

A severe affection of the pleura will but rarely occur, principally because the course of the disease is a very rapid one. [Not corroborated by Steele's statistics.—Ed.] If the duration is protracted an emphysema may follow. The temperature does not show any characteristic changes. In grave cases, which rapidly terminate fatally, fever may not be present at all. I have observed cases of carbolic-acid poisoning in which it was entirely absent. If it does set in, its course is absolutely irregular.

The following complications occur: Fresh warty vegetations on the heart, which Nauwerck observed in one of his cases of aspiration pneumonia after ether narcosis; icterus, mentioned by Silbermann; albuminuria, observed by myself.

The blood of children affected by septic pneumonia showed, according to Silbermann, marked abundance of the white, and extreme disintegration and dissolution of the red blood-corpuscles. Deep ulcerations in the larynx, which led to perichondritis of the arytenoid cartilage, were demonstrated in the above-described case.

DIAGNOSIS.

In the first place, the differentiation between aspiration and catarrhal pneumonia, both in an anatomic as well as in a clinical sense, has to be taken into consideration. It is true that at the onset the objective signs of the two affections do not differ materially; in either case it is only necessary that signs of numerous areas of consolidation in the lower lobes be present, consequently a decrease of the percussion sound with or without tympanitic accessory sounds; later on more or less extended dulness and, on auscultation, râles and sometimes bronchial respiration are noted. During the further course of the disease aspiration pneumonia is complicated by abscesses or gangrene, while these occurrences as consequences of catarrhal pneumonia as yet lack confirmation. In several statements regarding the appearance of abscesses in the train of catarrhal pneumonia, I think a confusion of the latter with aspiration pneumonia probably occurred. It is scarcely to be assumed that purulent bronchitis, which may complicate catarrhal pneumonia and cause numerous purulent areas to appear on the cut surface of the lungs, may be taken for an abscess.

The differentiation of the two forms of inflammation will be the less difficult the more carefully the causal factors are taken into consideration. Catarrhal pneumonia is far more frequently a disease of childhood, only that occurring in the train of influenza (page 560) being an exception to this rule; while aspiration pneumonia, in keeping with the conditions of its origin, is a disease that appears mostly in mature age. It is true that it will have to be investigated whether the pulmonary diseases occurring in children in connection with diphtheria are to be regarded as aspiration pneumonia, principally and exclusively, as is done by Henoch.

Accordingly it has to be taken into consideration whether one of the above-mentioned causal conditions—a fall into the water, unconsciousness with tracheal râles—has preceded. Although in most cases the importance of such a striking occurrence prevents us from mistaking it for the origin of the disease, yet there are instances, like the one described on page 623 in which the etiologic connection is likely to be overlooked. Possibly the sphere of aspiration pneumonia may be enlarged in the future, and then many other diseases may be included in the same category. It is not improbable that children who have become weakened as a consequence of gastro-intestinal catarrh may occasionally, when vomiting, aspirate a part of the food, and thus give rise to a cause for an inflammatory change.

If there is disease of one lung accompanied by tissue destruction, a regular examination of the sound lung will be necessary to determine the appearance of pathologic changes which cannot very well be taken for anything but a consequence of aspirated material. The condition is similar in perforating abscesses or neoplasms from organs in the neighborhood of the lung.

In cases in which the process originates from the aspiration of inflammatory material from tuberculous areas, according to Bäumler, an important diagnostic point is the sudden appearance of hemoptysis, which is almost immediately, or within a few days, followed by rapidly increasing fever and diffuse physical signs in the finest bronchi, with rapidly progressing dyspnea and cyanosis. This variety is distinguished from miliary tuberculosis by a more rapid course, higher fever, more quickly arising dyspnea, and the pronounced and rapidly increasing physical signs in the chest. They consist in numerous rhonchi, the early appearance of fine and crepitant râles, as well as early changes of the percussion sound, although the latter may take place only in small areas.

PROGNOSIS AND COURSE.

The chances for recovery depend principally upon the causes of the disease. Whenever aspiration pneumonia sets in, in connection with already existing affections, the aspirated material is usually infectious, whether it be a question of diphtheria, perforating abscess, new-formation, or gangrene of one lung. This is aggravated by the fact that the supply of infectious material is slowly exhausted.

The prognosis is most favorable in persons who, after a fall in the water, aspirate nothing but water instead of air. The water in most instances does not contain any, or only very few, infectious germs. In spite of fluids being demonstrated in the bronchi by an objective examination, inflammatory changes need not necessarily develop, and, even if they do occur, they may soon disappear again. The result is more doubtful if the fluids contain pathogenic germs, as in the cases described, in which newborn infants intrapartum aspirated the fluids from the mothers who were attacked by puerperal fever, or in the case of a fall into a sewer, which I shall report later on.

The chances for recovery are uncertain in cases in which saliva mixed with tracheal mucus entered the bronchi and the alveoli, the patient being in a state of unconsciousness. The duration of unconsciousness, and hence the quantity of the inspired material, are of essential importance in this case. As a rule, the course is a very rapid one, whether it end in recovery or death. A case like the following is probably exceptional:

A laborer, Albert H., thirty-seven years old, previously healthy, as was ascertained later on, on the evening of June 4, 1896, when loading manure fell into the pit, which was filled with putrid matter. He was taken to the hospital soon after, completely unconscious, with small pulse and stertorous breathing. The whole body was covered with filth. Eyes and lips had to be cleaned. The patient did not respond when spoken to. After an injection of 8 gm. (f℥ij) of camphor solution (6 camphor to 24 ol. olivarum) and a tepid bath with cold douches, the pulse became more quiet, the respiration slower. At the same time the patient was completely confused, tossed about, even tried to leave the bed. Half an hour later he showed maniacal unrest and his pulse became smaller again. Another injection was made of 4 dcg. (♏vj) camphor,—*i. e.*, the contents of a syringe which holds 2 gm. (f℥ss) of the above-mentioned camphor solution,—and of 12 cg. (2 grains) morphin besides. After half an hour he went to sleep, and slept until morning. On waking he stared about, showed his tongue when asked to do so, but opposed an examination so forcibly that for the time it could not be made. Vomiting of undigested contents of the stomach followed.

The examination of the lungs, made on June 6th, showed only slightly tympanitic percussion sounds at the left base posteriorly, but the respiration was purely vesicular. In addition, severe conjunctivitis and violent catarrh were to be noticed. No fever. Urine free of albumin. Patient was perfectly conscious, but could not give any information in reference to his accident.

Next day, in spite of dissuasion, he demanded to be discharged from the Altstadt Hospital. Three months later the Neustadt Hospital transferred him back to the Altstadt again. Two days after his discharge from here he had to ask for admission in the other hospital, because violent cough and fever had set in.

On September 14, 1896, he was very low. His debility was so great that he was not able to stand or to walk; his complexion was very pale. The liver reached almost to the umbilicus, the hepatic region was very sensitive to pressure. Dulness and ringing râles in the right supraclavicular and infraclavicular region; from here on, absolute dulness. Auscultation showed, right anteriorly, cavernous respiration and râles all over the chest. Right posterior dulness from apex to base; slow, almost abolished, respiration. Pectoral fremitus was not weaker on the right than on the left. Puncture with Pravaz syringe proved pus in the thoracic cavity which was discharged by resection of the ribs under chloroform narcosis. The pus amounted to more than 500 c.c. and emitted an intensely gangrenous odor. After the pus, free of any blood, had all been evacuated and the patient awoke from his narcosis, a violent hemorrhage from the pulmonary tissue set in. The blood soon oozed through the full width of the cut. The pleural cavity was at once packed with a long strip of iodoform gauze which had filled at least 500 c.c. of a test tube. The hemorrhages ceased, no blood was discharged through the bronchi. In the evening pulse, temperature, and general condition were quite satisfactory.

On September 16, 1896, a collapse occurred with cold perspiration which soon passed away after subcutaneous injection of camphor oil and the administration of stimulants.

On September 17, 1896, the packing was carefully removed from the thoracic cavity; no hemorrhage; no fever.

September 26, 1986: Dulness in right supraclavicular and infraclavicular region, bronchial respiration, and râles; right supraspinal dulness. The weight of the patient 48 kilograms.

October 25, 1896: In spite of continuous cough the general state of health had improved. No more discharge from the place of resection. With violent cough some little blood was expectorated. Sputum did not contain any tubercle bacilli. Weight increased to 57.5 kilograms.

November 7, 1896: The incision had entirely closed. In the right anterior region up to the third rib, hollow percussion sound, bronchial respiration, and ringing râles. In the right supraspinous region, dulness, bronchial respiration, and râles; from here on, loud percussion sounds and vesicular respiration. The left lung during the entire courseshowed normal conditions. Elastic fibers were found in the sputum, but no tubercle bacilli. Weight 60 kilograms.

March 10, 1897: The subjective state of health of the patient and the objective conditions had steadily improved. He looked well and his weight had increased to 68 kilograms. Cough and expectoration still existed, but were only moderate. Tubercle bacilli were not found upon repeated examinations. In the right anterior region, up to the third rib, the percussion sound is still hollow and high, but the inspiratory murmur is vesicular; the expiratory murmur prolonged. Dulness over the right fossa supraspinata; inspiration coarse, expiration prolonged. In consideration of his private affairs the patient desires to be discharged from the hospital.

The above communication justifies the conclusion that aspiration of fetid material took place when the patient fell into the pit. The entire condition of the man when first brought to the hospital—the filth on eyes and lips, the stertorous breathing—does not admit of any doubt of such an occurrence. The phenomena of disease which then appeared in the heretofore healthy man, and which were principally dependent upon the gangrenous disintegration of the pulmonary tissue and the ensuing empyema, can only be traced to lobular areas produced by aspirated material; while a tuberculosis, the only other process to be taken into consideration, can with certainty be excluded on account of the left lung remaining exempt; the non-appearance of tubercle bacilli; and the extraordinary increase in weight from 48 to 68 kilograms.

PROPHYLAXIS AND TREATMENT.

The prevention of aspiration pneumonia can only be brought about in cases in which fluids or the contents of the mouth have entered the lungs. In every instance unconsciousness is present, and the first duty is to shorten this condition as much as possible. In individuals who are taken from the water and are unconscious, artificial respiratory movements must be undertaken forthwith, and all other means may be tried for resuscitation, and for removal of the fluid from the bronchi. In the case of unconsciousness as a consequence of carbonic oxid poisoning, fresh air, a tepid bath with cold douches, and subcutaneous injections of camphor solution will prove advantageous. In carbolic-acid poisoning in consequence of having swallowed carbolic acid, the stomach must be washed out until the contents cease to smell of carbolic acid. Complete recovery was secured in a man who, after inadvertently drinking carbolic acid, was, luckily, brought to the hospital within half an hour after the event, and in whom lavage was used. In this case also a concentrated solution of sodium sulphate (10 gm.—f℥ijss—of a 1% solution) was given subcutaneously. The latter was likewise applied in another case in which a nurse by mistake injected a liter of a 2½% solution of carbolic acid into the intestine. The latter case was explicitly described by Friedberg.

In treating a manifest aspiration pneumonia the possibility of an injurious effect of aspirated pathogenic germs is to be avoided in the first place. The most rational way is the inhalation of carbolic acid in a 4% solution. In the usual application with steam atomizers this solution is still more reduced. Later, to control the cough which is always present if the disease last some time narcotics should be used and, if necessary, expectorants. Other sequels, as gangrene and empyema, are to be treated according to their nature.

DESQUAMATIVE PNEUMONIA.

By the name of "genuine desquamative pneumonia" Buhl has designated a special form of pulmonary disease which he was the first explicitly to describe. His communication, however, has not been fully appreciated until now. Why this should be so, I can only partly account for, as there is no more appropriate designation for this variety of pneumonia, which must not at all be taken to be a rare pathologic-anatomic curiosity, but, on account of its frequency, deserves special clinical treatment and exhaustive description.

For the correctness of this view an example may be mentioned. A young man about twenty years of age, and apparently in the best of health, was suddenly attacked by hemoptysis. In percussion of the pulmonary apices, which, with some caution, may be done without detriment, no dulness was found anywhere. Only on auscultation catarrh of one apex could be made out, and by this means the seat of the affection located. If the chest is examined two weeks after the occurrence of hemoptysis, complete dulness over the upper lobe may be found, appearing without any particular symptoms. Neither cough, pains, nor fever need give a hint that the hemoptysis has been followed by the development of an extensive dulness of the upper lobe. After having lasted for weeks this dulness may disappear and the affected upper lobe become normal again.

There is no doubt about the actual appearance of such a pathologic change after hemoptysis. Every physician must have made similar observations. It seems, however, as though generally but little attention is paid to the pathogenesis of this condition. Possibly this is due to the fact that the onset and the disappearance of the change cannot be demonstrated until quite a time has elapsed, consequently such observations can better be made by a practitioner in his private practice than by the physician in a hospital; and the former has not the time nor the leisure to report his observations for the general benefit of the profession.

Without any doubt in such cases it is a question of a peculiar pneumonic consolidation in which resolution may occur. This peculiarity, however, is best characterized by the designation "desquamative pneumonia" chosen by Buhl, as will be demonstrated in the following description.

The above remark, that the failure to admit the actual occurrence of this form of disease as defined by Buhl is only partially explicable, is based on my opinion that Buhl ascribed too great an importance to this affection. He intended "to enforce a new view, to bring genuine desquamative pneumonia into prominence as a separate disease, even if appearing in several stages, and accuse it of being the primary stage and the accompaniment of most important processes, dangerous to life,—pulmonary phthisis and tuberculosis; he wanted to connect it genetically with these already well-known conditions," and has exhaustively described the process by which desquamative pneumonia leads to chronic fatty degeneration, to cirrhosis of the lungs, and to caseous pneumonia. Two stages should be distinguished here—an acute, and a subacute and chronic stage. In the former the lungs are enlarged in volume and weight, they show on the cut surface the striking red porphyry-appearance in which on a red ground large and small, whitish-yellow, isolated and agminated granules are seen. These whitish-yellow deposits are either isolated granules of the size of the smallest gravel, or several such granules unite to form conglomerations and compact masses as large as a pea and larger, of irregular, ill-defined contours. They are yellow, dry, decayed, airless and bloodless tissue-parts which on account of their rigidity do not sink into the cut surface, but project. According to size they correspond to either single pulmonary vesicles or more frequently to entire pulmonary lobules, or a bundle of lobules varying in size, or even to the greater part of an entire lobe; but they are rarely sharply defined and gradually merge with the red background. Examined microscopically, the yellow deposits dissolve into shrunken alveolar epithelia or into their remnants (disintegrated nuclei and nucleoli) and into the necrotic stroma; for this reason fat-granules are found in rows, interspersed in the connective tissue and in the membranes of the vessels.

In the subacute and chronic stage the whitish-yellow anemic eschar areas after they have grown to the size of a pea and larger remain stationary, while the red ground, the desquamative, inflamed part, may be quickly and completely brought back to the normal condition, after transitory fatty degeneration.

According to Buhl, this is the first possible issue which is the more apt to appear the smaller the number of yellow areas and the farther they are apart. The lungs, which are otherwise perfectly healthy, contain in this case multiple areas of caseous tissue which plainly surrounds a bronchiole.

Another termination which may take place in the acute stage of caseous pneumonia leads to cavity formation. That is to say, if the process is very intense and the eschar formation extends in "uninterrupted foci" over a considerable portion of the lung, while the individual foci are rarely smaller than a hazelnut, the entire necrotic mass, the entire focus, may resemble a gangrenous plug by demarcated suppuration, separate, and fall off; so that a loss of substance in the pulmonary parenchyma as large as the necrotic part may result.

In giving this description of the entire genesis of the disease, which is essentially based upon pathologic findings, Buhl, according to my opinion, proceeded without the aid of clinical observation. The latter proves conclusively that desquamative pneumonia is a rare occurrence as a primary disease in a previously healthy respiratory organ, whereas it appears frequently as an accompaniment of an already existing disease of the pulmonary apices, and is then the cause of further focal disintegration of the pulmonary tissue. Buhl's error may be traced to the fact that he took the desquamative pneumonia which occurs in connection with already existing foci in the pulmonary apices, and frequently affects the entire upper lobe, for a disease beginning primarily in normal lungs; and regarded the already existing apical affection, which often runs its course without producing symptoms and may be restricted to a small area, for a consequence of desquamative pneumonia, while the reverse is the correct view.

Because not only in connection with hemoptysis, as mentioned in the beginning, but also in connection with small tuberculous areas, an extensive desquamative pneumonia may take place, the course of which is of three different kinds: (1) A *restitutio ad integrum* may occur; (2) a chronic pneumonia may follow, as will be explained later on, when describing this form; and (3), exactly as Buhl states, small caseous areas may appear in the affected portions which undergo necrosis, so that cavities take their place.

These different terminations of chronic tuberculosis of the lungs have long been known. We designate them more empirically (1) as complete disappearance of extensive consolidations over the upper lobe with more or less absolute recovery; (2) as extensive slaty induration around smaller cavities in the pulmonary apices; (3) as a focal disintegration of the pulmonary tissue in connection with or without hemoptysis and following an already existing area or small cavities in the pulmonary apices with a mostly acute feverish course.

If we do not want to forego every genetic explanation of these occurrences and leave their entire clinical course unenlightened, we must recur to desquamative pneumonia.

For better proof, nay, in order to compel the acknowledgment of the importance of this disease, in spite of the example given at the commencement of this section, I may ask: Has not every carefully observing physician already had the opportunity of ascertaining in connection with an apparently unforeseen hemoptysis, and also without it, the presence of a consolidation of an upper lobe, and likewise frequently seen it completely disappear after strict observance of appropriate hygienic, dietetic, and therapeutic measures? How else can this consolidation be explained but by a pneumonic consolidation, which, from the beginning, leaves the pulmonary tissue intact and only affects the pulmonary alveoli or their epithelial lining, and which may fully disappear by resolution, so that the entire affected portion of the lung becomes normal again? In the autopsy of individuals who died with all the symptoms of phthisis, which of course includes the finding of tubercle bacilli, who has found, in addition to hazelnut- to walnut-sized cavities, a firm uniform fibrous consolidation of the rest of the upper lobe, and who will claim this to be anything but the result of chronic pneumonia? Who has not seen an originally uniform consolidation of one lobe, generally the right upper one, from which later on, with expectoration of sputa containing tubercle bacilli and disintegration of tissue within the affected section of the lung, there developed the characteristic percutory and auscultatory signs?

A uniform explanation of all these processes is only possible by the conception of desquamative pneumonia. We will leave it undecided regarding the character of the disease whether Buhl is right in saying that "the disease, like genuine Bright's disease, myocarditis, acute hepatic atrophy, is not a mere concomitant, but the expression of a general disease localized in the lungs." Considering the cogent fact that pulmonary phthisis with its self-evident criterion, the tubercle bacillus, is a very frequent termination of desquamative pneumonia, I confine myself to a remark regarding the share of the tubercle bacillus in the entire process. If those authors were right who declare tuberculosis of the lungs to be a strictly infectious disease, in which the tubercle bacillus settles in the normal pulmonary tissue, then the above referred to heterogeneity of the issue of desquamative pneumonia—(1) complete recovery, (2) chronic pneumonia, (3) tuberculous (bacillary) disintegration—would be inexplicable.

If, however, we assume that the tubercle bacillus can settle only in diseased lungs, the entire process becomes comprehensible. It is to be regretted that those authors who regard pulmonary tuberculosis as a strictly infectious disease do not sufficiently consider the pathologic anatomy of pulmonary tuberculosis. I need not go into details here regarding the relations of Bacillus tuberculosis to the origin of pulmonary phthisis.

PATHOLOGIC ANATOMY.

Buhl describes the macroscopic condition of the lungs in desquamative pneumonia in the following appropriate manner: "The disease may be limited to one lobe, which is generally the upper lobe. Or it attacks one, but the entire lung; this is more frequent, and then the upper lobe is generally affected more than the lower lobe. Or it attacks both lungs only in the upper or in all lobes, and in this case one lung is mostly more intensively affected than the other. But almost invariably the process is more developed in the upper parts, and evidently progresses from above downward, although in very acute cases all parts seem to be attacked at once. Very rarely the same degree and the same duration can be observed in the different lobes. In cases of from six to eight weeks' duration the volume and weight of the diseased lung or lobe are materially increased; the surface is smooth and glistening; the pleura is swollen, and here and there, especially over the lower lobe, the lung is covered with ecchymoses. The parenchyma does not collapse, neither in taking out the lungs nor after section; the elasticity of the parenchyma appears to be suspended or impeded; the tissue remains rigid. In spite of this the brittleness seems to have increased, and continues to increase with the duration of the affection. The cut surface shows the lobar diffuse extension of the affection, more or less diminution, at times even absence of air and feeble granulation. According to the duration of the process, the layers of granular pigment increase. The color of the tissue becomes slate-gray, even black, in consequence. In many cases the brittleness becomes so pronounced that an actual diffuse softening takes place which may involve the greater part of a lobe."

In connection with this description of Buhl, I have to emphasize the fact that desquamative pneumonia may not only originate in the upper lobe, but can also first appear in a lower lobe, infect the upper lobe of the same side, and then attack the other lung. I myself made such an observation, and I believe I may include a case of Heitler's in this category. I shall refer to this case later on.

Rare as is desquamative pneumonia in the lower lobes, it is equally rare for the disease to develop in a previously healthy lung, or one the apex of which merely contains old fibroid areas of small extent, when the apex of the other lung is the seat of tuberculous infiltrations or cavity formation.

Much more frequent, however, is the consecutive form of desquamative pneumonia. As such I think I may designate the affection which appears in connection with already present tuberculous infiltrations and excavations at the pulmonary apices. If it can be observed in an earlier stage on the dissecting table, the absolutely airless upper lobe shows on the cut surface with a firm consistency a uniformly gelatinous or gray aspect with interspersed yellowish-gray areas. Here and there single soft places may be found, from the size of a bean to that of a hazelnut, rarely larger, which without marked demarcation gradually disappear in the surrounding tissue. Not until in the later stage, when the areas have changed into cavities, a marked border separates them from the surrounding tissue, which may have become normal again or may have undergone fibroid thickening.

I do not think it necessary to enter upon the somewhat aphoristic results, which are reported by Buhl, of the microscopic examination, because they will give little information. A case, however, which is described by Heitler deserves special mention. In a man who died with the characteristic symptoms of genuine pneumonia, whose right lower and middle lobe were in a state of complete hepatization, the microscopic examination gave the following results:

> The contents of the alveoli were principally large formed elements which had to be taken for changed alveolar epithelia; they appeared free in the lumen of the alveoli, only seldom embedded in a filamentous or crumbling mass, and either isolated, distributed in the lumen of the alveoli, or united in groups of up to eight, at times in such a way that they completely filled an alveolus; the form was partly angular, or more or less rounded, or entirely round, the contents finely granular with two to three large nuclei; others showed endogenous forms. Some alveoli still contained remnants of red blood-corpuscles; the alveolar walls were bare and but rarely traversed by distended vessels.

Heitler remarks with reference to the above:

> The most surprising condition in this result was the not absolute absence, but only very small number, of pus corpuscles in the alveoli and the large quantity of large formed elements—alveolar epithelia in their different stages of metamorphosis—in them. To meet every doubt, one has to ask the question whether in this case an early stage of pneumonia may not have been present, possibly of beginning infiltration, in which we also find large changed epithelia in the alveoli. Without reference to the fact that the lung macroscopically showed the typical picture of gray infiltration, the other signs of beginning infiltration were wanting microscropically also—namely, the enormously distended vessels and the hemorrhages; clinically we had the symptoms of a complete infiltration. In view of all this, nothing remains but to presume an inflammatory process of the lungs which does not fit the pattern of common croupous pneumonia, although clinically it could not be distinguished from the same. The character of the process consists principally in a desquamation and multiplication of the alveolar epithelia; we could, therefore, speak of a strictly desquamative pneumonia, had this name not been used to designate another process in the lungs; in regard to the other symptoms also this case has nothing whatever in common with genuine desquamative pneumonia as described by Buhl, so that a comparison and connection of the two processes cannot be thought of.

If I do not quite agree with this opinion, I may vindicate my contradiction by saying that, according to my idea, the author did not feel inclined, in consideration of his purely casuistic communication, to enter upon a criticism of several untenable points in Buhl's views. After my modification of Buhl's somewhat extravagant conception, of the importance and consequences of this disease, I think I may include the case of Heitler, because in all cases of desquamative pneumonia which I had a chance to examine at so early a stage I found almost identical changes.

The microscopic appearance of the pulmonary parenchyma in the earliest stages of the disease is absolutely surprising by the uniformity of the change. It manifests itself by a complete filling of the alveoli with swollen epithelia. There cannot be a moment's doubt that it is a question of epithelia. Large cells with large nuclei are lying close together. There is no space between them and the alveolar wall; their protoplasm mostly has a light appearance. One or two small nuclear bodies are seen in the nuclei. But there is hardly an alveolus present in which there is not also another kind of cell which as a rule greatly exceeds the light cells in size, but has quite a dark appearance, caused by a complete filling of the cell with fat-globules of fairly uniform size.

Quite a number of the cells with the bright protoplasm contain two or more nuclei. There are some cells which contain as many as five large clear nuclei, and therefore are four and more times larger than those containing one or two nuclei. They deserve the name "giant cells." With reference to the explanation on page 397, I may ascribe their origin to the coalescence of single alveolar epithelia.

I must leave it undecided whether the remarkable circumstance that only a small number of larger fatty epithelia appear, and it never happens that all the epithelia undergo fatty degeneration, is to be accounted for by the fact that under normal conditions plates without nuclei exist, and that those cells which turn into such plates are the first to undergo fatty degeneration in this disease.

Very rarely single red blood-corpuscles are found free in the lumen of the alveoli at this time.

All the capillary vessels of the alveoli are completely filled with blood-corpuscles. But it seems to me that the engorgement is not so excessive, that the capillaries are not so distended, as in croupous pneumonia.

In the further course a total desquamation of the epithelia sets in, both of the distended cells, those with one or more nuclei, and of the degenerated ones.

One case of desquamative pneumonia of the right upper lobe with previous consolidation of the left pulmonary apex was especially instructive to me. The case was that of a man seventy-two years old. The left pulmonary apex was firmly adherent to the thoracic wall. On separating the same, the decayed tissue tore into a ragged mass, which was airless and slaty. The upper lobe of the right lung was large, heavy, and airless. The upper third of its cut surface had a pink, hyaline appearance, and not very great consistency. The other larger part of the upper lobe was grayish-white, firm, and showed single blackish points. Toward the center an area of the tissue the size of a plum was markedly decayed.

The microscopic examination of the apex showed only a part of the alveoli to be filled with distended epithelia; a very large number were without any cellular contents. In preparations which, for the purpose of staining, were repeatedly washed with water and then with alcohol, every one of the alveoli was without cells. This does not necessarily imply that such had been the case during life, but it proves mainly, in comparison with specimens from other kinds of pneumonia, that the epithelia adhere much less firmly to the wall.

These parts which are destitute of any epithelial elements offered an exceedingly favorable opportunity to examine the conduct of the pulmonary supporting tissue. It proved to be perfectly intact; rarely a more favorable opportunity will offer to demonstrate the normal conduct of the elastic tissue of the lungs and its relations to the vessels and capillaries so distinctly as just in this stage of desquamative pneumonia.

The examination of the ensuing stage gives a better confirmation of the total desquamation of the alveolar epithelia taking place in the course of the disease. This stage can be observed more frequently than the just-described first stage. Only in the case just mentioned I found both in one upper lobe. The far larger lower part showed a condition which was entirely different from that of the upper third. The alveoli in the entire lower part were filled with an extremely regular fibrinous network which contained nothing but small round cells—leucocytes. No alveolar epithelia were anywhere to be seen. Here and there single fibrinous threads connected the contents of neighboring alveoli, through the wall of which they extended, exactly as occurs in croupous pneumonia. In numerous alveoli the leucocytes were comparatively few, not all the fibrinous meshes were filled by them; in some places they were more dense and covered the fibrin entirely. The latter was missing throughout in those parts which were nearest to the softened area; leucocytes alone formed the contents of the alveoli, until finally in the softened parts, as far as they could be examined, the contents of the alveoli became entirely confluent. Wherever a boundary was still in existence between the single alveoli, it was no more indicated by the elastic tissue, but by the fine interalveolar vessels copiously filled with blood.

In another case, of a man forty years of age, I found results deviating from the normal course as here described. The patient was brought to the hospital moribund; he had dulness over the right upper lobe and hemorrhagic sputa. He was not able to communicate with any one. Death occurred three hours after he reached the hospital. At the autopsy a cavity the size of a hen's egg was found in the left upper lobe, and near by several caseous areas. The right upper lobe was large, heavy, and uniformly firm. Diffuse congestions appeared on the cut surface on a uniform grayish-white ground. The microscopic examination showed a complete, dense filling of the alveoli with round cells between which only occasionally the described fibrinous net was visible. Besides white blood-corpuscles, numerous red ones were found in many places; blood was found not only in the alveoli, but also in their interstices and in the adventitial tissue of the smaller bronchi.

The fact that the alveoli throughout the entire diseased lobe were filled with white blood-corpuscles, while the hemorrhage was confined to single parts, although in fairly uniform distribution, and the fact that in these places also most of the material contained in the alveoli consisted of white blood-corpuscles, allow the inference that the hemorrhage did not take place until after the exudation of lymphoid cells into the alveoli. In these hemorrhagic parts the nuclei of the white blood-corpuscles could not be demonstrated by staining.

By reason of these results the genesis of desquamative pneumonia may be described in the following manner:

The disease commences with a swelling of the alveolar epithelium, with synchronous increase of the nuclei, and frequent coalescence of the cells to form giant cells. A part of the epithelium undergoes fatty degeneration with considerable increase in volume. Then follows a desquamation of the cells of the walls of the alveoli, so that later on—apparently in cases with unfavorable termination—the entire epithelium is lost. Next an exudation of fibrin and white blood-corpuscles takes place. This process shows a material difference from the course in croupous pneumonia, in which the exudation of white blood-corpuscles is preceded by a more or less considerable hemorrhage. The fibrin decreases with the increase of the white blood-corpuscles; as in suppuration, softening of the pulmonary tissue proper follows, mostly in circumscribed areas, and this softening is followed by the appearance of cavities. If a hemorrhage complicates it, it does not set in until the complete filling of the alveoli with white blood-corpuscles; it is but a result of a disintegration of the vessels caused by the action of these cells. This course is analogous to the process in a hemorrhagic inflammation of other organs, and does not correspond with that of croupous pneumonia, in which hemorrhages set in immediately after the affection of the alveolar epithelia, representing a kind of thrombus (see page 402) into which white blood-corpuscles do not enter until later on.

SYMPTOMS.

Primary desquamative pneumonia generally sets in in the same manner as genuine pneumonia—namely, with chills. The temperature rises to a considerable height, but does not show a strictly remittent type. The changes which can be demonstrated by auscultation and percussion also show all the signs of genuine pneumonia. The sputa, according to Buhl's observation, are also similar in character. In Heitler's case they were likewise rusty, but otherwise did not show anything remarkable. According to my very limited experience in the clinical sphere,—because the majority of cases of primary desquamative pneumonia I only recognized as such on the dissecting table,—the sputa were not rusty, except in the above case, which was not brought to the hospital until a short time before death. In a case originating in the right lower lobe sputa were absolutely wanting in the first three weeks.

Buhl names a special characteristic of the sputum which is said to be infallible for the diagnosis of desquamative pneumonia. He says: "Even during the first week, when the fever, the crepitant râles heard all over the chest, the somewhat empty and sometimes distinctly metallic percussion note, the bronchovesicular or even bronchial breathing, and the bloody expectoration combine to cause the disease to be mistaken for croupous pneumonia, microscopic examination of the sputa yields positive signs that the disease is neither catarrhal nor croupous, but desquamative pneumonia; because neither in catarrhal nor in croupous pneumonia is alveolar epithelium found in such quantities in the sputum, which, besides, never contains ciliated cells. The longer the disease lasts, the more epithelia and granular cells are found, the more the fatty degeneration has advanced, the sooner the cells contain pigment. Free molecules of fat, free cell nuclei resulting from disintegration, are of course also present."

In the case I observed, in which desquamative pneumonia originated in the right lower lobe, nothing but a little mucus was expectorated during the first three weeks. This did not contain any tubercle bacilli. Not until the fifth week of the disease were numerous sputa thrown out in which tubercle bacilli could be demonstrated.

The symptoms are quite different when a consecutive desquamative pneumonia of the upper lobe is added to an existing small focus in the apex which so far has not given rise to any pulmonary symptoms. The desquamative pneumonia is mostly preceded by hemoptysis, which I consider to be a proof that an already existing focus in the pulmonary apex has separated from its surroundings, thus causing a rupture of smaller vessels. Not until a few days after this occurrence the change in the upper lobe takes place. The percussion sound becomes empty and high-pitched or dull; the respiration slow, weakened, not rarely bronchial; fever need not necessarily be present.

The subsequent course varies. The dulness as well as the abnormal respiratory sound may disappear entirely and a *restitutio ad integrum* may occur. The case of a young lawyer who consulted me on account of a grave anemia is very remarkable. He was not bedridden and did not show any symptoms relating to the lungs. I found a desquamative pneumonia of the entire right upper lobe. An absolute disappearance of all objective symptoms was brought about by an interruption of his business activity, and sojourn in a bathing-place on the sea. A year later he had a hemorrhage. I now found a tuberculous affection of his right lung, which six months later ended in death. Just as hemoptysis may be succeeded by desquamative pneumonia, the latter may also occasionally take place without it in connection with an already existing tuberculous area in the pulmonary apex. The onset of desquamative pneumonia may be so gradual that the first examination may divulge an extended infiltration. The disappearance of such an infiltration may have caused many erroneous views in regard to the value of therapeutic methods.

I must leave it undecided whether the following condition may be regarded as caused by primary desquamative pneumonia or whether they occur in connection with smaller areas at the apex, and must, therefore, be considered as secondary—namely, consolidation in one or both upper lobes after preceding cough, which I have observed in children between four and ten years of age. The cases were not accompanied by fever, and with the necessary precautions almost always terminated favorably after a moderately long duration.

By recognizing these desquamative pneumonic changes of the pulmonary upper lobes I have frequently been able to give the proper diagnosis for an otherwise incomprehensible process, when in consultation with other physicians. The point is that with proper treatment in some patients the dulness over the upper lobe disappears completely after some time, but can be demonstrated again after several weeks or months. If an explanation of these occurrences is possible at all, it must be based on the actual cure of a consecutive desquamative pneumonia, one that runs its course in the alveolar epithelium, and on the possibility of recurrence in the same place where it had existed before.

DIAGNOSIS AND PROGNOSIS.

The discrimination between primary desquamative and genuine pneumonia is of especial importance, but at present it still encounters great difficulties. Quite frequently primary desquamative pneumonia may be taken to be croupous, and the origin of grave consecutive conditions, principally of tuberculosis, may be laid to the door of croupous pneumonia, especially when the disease is located in the upper lobe; and, vice versa, it happens in rapidly occurring fatal termination that a pneumonia which has been taken to be croupous before life was extinct, on the dissecting table turns out to be desquamative pneumonia.

It will be necessary to have recourse to a minute microscopic and bacteriologic examination of the sputa, which so far has possibly rarely been done. Before all, it has to be demonstrated whether Buhl's view is correct, that the presence in the sputum of numerous alveolar epithelia is characteristic of this affection. Up to the present I have not sufficient foundation for this view. It is also possible that the absence of Diplococcus pneumoniæ in the sputa or its presence in small numbers is a criterion for the diagnosis if the other objective and subjective symptoms of desquamative pneumonia are present.

The diagnosis of consecutive desquamative pneumonia is less difficult in most cases. It is then only a question of demonstrating a more or less extended dulness over the upper lobe developing after hemoptysis or an already present tubercular affection of the pulmonary apex.

But if a tubercular area has existed in the pulmonary apex up to the time when an extended consolidation of the upper lobe can be demonstrated as apparently the first morbid change, then the diagnosis is apt to offer some difficulties. In the first place, extensive tuberculous infiltration of the upper lobe with disintegration of tissue must be excluded. This is indicated by the longer duration of the process, the intensity of the cough, the wide-spread crepitant râles, the tympanitic element in the dulness, the presence of metallic râles; while desquamative pneumonia, as long as it is still limited to the alveolar lumen, appears with little or entirely without cough, the percussion note is uniformly empty or dull, the respiration is weakened or faintly bronchial, and no catarrhal sounds or only such as are limited to a small area—namely, the previously present small tuberculous area which so far had been latent—are heard.

Likewise attention is to be directed to the differentiation of acute tuberculosis, in which, in connection with already present small areas at the apex, at least the upper lobes of the lungs, sometimes the entire lungs, are interspersed with acinous foci. This form never shows a uniform consolidation, limited, at least at the onset, to the upper lobe, and is attended with considerable fever, while consecutive desquamative pneumonia quite frequently takes place without fever, and patients often enough show so few disturbances of the general state of health that they are not bedridden at all.

The diagnosis of these conditions is the more important, as the prognosis in consecutive desquamative pneumonia is much more favorable and complete recovery may be looked for.

It is very difficult to state at present what the prognosis of primary desquamative pneumonia is. Many cases may terminate in recovery; but they may not have been recognized, being mistaken for genuine pneumonia. The termination may therefore possibly not be as unfavorable as seems to be the case at present, owing to the fact that the disease is mostly recognized on the dissecting table. Very likely here, as well as in the consecutive form, a *restitutio ad integrum* may take place; in other cases a focal disintegration may lead to the formation of tuberculous areas and a chronic consolidation may occur around these areas.

TREATMENT.

At present no special therapeutic indications can be given for primary desquamative pneumonia. In general, the precautions which are recommended in the treatment of croupous pneumonia may be observed, and a stimulating treatment should be instituted from the beginning.

In the treatment of consecutive desquamative pneumonia special therapeutic measures can be taken. Numerous observations entitle me to say that a favorable result may generally be looked for in such cases. Often enough I have seen extensive consolidations over an upper lobe, with weakened respiration or slight bronchial breathing, completely disappear.

In all cases, mostly appearing and terminating without fever, the business occupation or, according to the age, the attending of school has to be suspended. Whenever it could be done, I recommended sojourn in localities with good air, in a richly wooded neighborhood. Exercise, mountain-climbing, bicycle-riding, etc., were prohibited.

Special stress must be laid on an invigorating diet; above all, plenty of milk. In regard to drugs, I use quinin and iron in every case. For adults I prescribe:

℞. Quinin hydrochlorate .... 1.0 (15 grains)
Iron sulphate .... 3.0 (45 grains)
Powdered licorice root .... 3.5 (52 grains)
Extract of licorice, enough to make xl pills.
M. Dust with licopodium powder.
Sig.—Take two pills twice a day.

For children:

℞. Quinin hydrochlorate
Iron sulphate .... of each 1.5 (22 grains)
Powdered licorice root .... 1.0 (15 grains)
Extract of licorice, enough to make xxx pills. M.
Sig.—Take two or three pills daily, according to the age.

I have rarely had to use expectorants. If necessary, adults received twice daily, or only in the evening, 0.3 (5 grains) of Dover's powder; children, from 0.01 to 0.015 ($\frac{1}{6}$ to $\frac{1}{4}$ grain).

SYPHILITIC PNEUMONIA.

PATHOGENESIS AND PATHOLOGIC ANATOMY.

In describing syphilitic inflammations of the lungs hereditary syphilitic diseases must be distinguished from the varieties caused by acquired syphilis, and in the latter three kinds are to be defined: (1) gumma, (2) changes in the connective tissue, and (3) parenchymatous changes.

Virchow was the first to describe a peculiar white hepatization of the lungs in still-born children, in whom the alveoli were crammed with exceedingly plentiful epithelium, sometimes filled with fat. Every particle of the lungs sank in water, but in spite of this the lungs could be fully inflated. F. Weber made a similar description under the same name without mentioning any connection with syphilis. The disease appears, according to his observations, in children which are born one to two months before term. The lungs were so large that after detaching the sternum hardly anything could be seen of the pericardium and several impressions of the ribs could be plainly seen on the lateral surfaces of the lungs. The color of these large lungs on the surface and on all cut surfaces which were uniformly smooth was almost white with a tinge of yellow, and the firmness of the anemic pulmonary parenchyma was so considerable that every single lobe stood like a tenpin when placed on a table. Herker, when describing such a case, in which the thymus gland was almost entirely changed into an abscess filled with straw-colored pus, and the pale red lungs, which filled the thorax as completely as lungs that have expanded, at the same time felt exceedingly firm on the cut surface, was the first to state that Merkel declared this change to be a rare form of chronic pneumonia peculiar to syphilis which originates *intra uterum* and is accompanied by increase of the epithelium and consolidation of the pulmonary cellular tissue.

Lorain and Robin give a very explicit description of two cases which should be counted as belonging to this category. They demonstrated in two children, born in the sixth and seventh month respectively, one of whom lived only long enough to breathe three times, while the other lived two hours, besides pemphigus, the pulmonary change which they compare with a case observed by Rogers and named by him "*pancréatisation du poumon.*" By reason of their microscopic examination they declare this affection to be characterized by a considerable increase of the pavement epithelium which fills and obliterates the bronchioles in the shape of cylinders. They accordingly designate the disease pulmonary epithelioma.

According to Wagner's investigation, not a trace of alveoli is found in the syphilomatous lungs of the newborn; only occasionally some alveoli are present which are four to six times smaller than those in children who have breathed. The interalveolar tissue is much increased in width by compact layers of medium-sized, round, free nuclei, and large round cells with large nuclei; in most places plenty of albumin and fat molecules are present, interspersed with atrophied or fatty degenerated nuclei and cells.

My own observations of syphilitic pneumonia of the newborn enable me to add some further details to this description. I had the opportunity of observing such cases in the lying-in department of the Altstadt Hospital, which was under my direction. Into this department the women about to be delivered were transferred from the syphilitic ward.

The majority of these children were fully mature, and some of them breathed for a comparatively long time;—one, three and a half hours; another, five hours. At the autopsy the lungs were found large, heavy, firm, airless, and of characteristically white appearance. Generally the tissue showed uniform consolidation on the cut surface; rarely—I only remember two cases—there was a peculiar small lobulation. The macroscopic appearance is aptly described by the designation "pancréatisation," which was undoubtedly chosen by Lorain and Robin exclusively for such cases, and which means that the condition corresponds on a smaller scale with the macroscopic structure of the pancreas.

In the microscopic examination I found the greater majority of the alveoli empty in every instance; only a small number were completely filled with well-preserved cells. But generally the empty alveolus was smaller throughout than in newborn children who had breathed. Most striking was the increase in the width of the alveolar interstices. This broadening was caused exclusively by swelling of the alveolar epithelium, which was firmly adherent to the wall.

Besides this, another important change had taken place in the smaller vessels. Here all parts of the wall showed an astonishing increase in thickness, affecting for the most part the adventitia, and to a lesser degree the muscularis (compare Plate 7, Fig. 8). Frequently the adventitia was of the same diameter, both in longitudinal and in transverse sections, as the lumen of the vessel. At different places the original substance appeared to be filled with hyaline material. Between the adventitia and the surrounding tissue an empty space was usually found which probably corresponds to the dilated lymph-space.

I believe I am entitled to bring these two changes into genetic connection and trace the thickening of the alveolar interstices in consequence of the distention of the alveolar epithelia to the excessive thickening of the walls of the blood-vessels. I came to this conclusion by the findings in cardiac lesion lungs which will be described later. Here, where congestion is the only cause of the pulmonary change, the conduct of the alveoli shows a striking resemblance to that in syphilitic pneumonia of the newborn. This similarity, and the consideration that such a thickening of the vascular walls as is present in these cases must affect the nutrition of the pulmonary tissue in much the same way as the congestion in the presence of cardiac lesions, induce me to declare the affection of the pulmonary tissue to be a consecutive condition of the vascular disease and consequent impaired function of the vascular walls.

While these changes are present in all cases of white syphilitic pneumonia, whether a uniformly smooth consolidation is present or whether the tissue is lobulated like the pancreas, I have also found in the latter form a conspicuous dilatation of all the finer bronchi, and I believe I may trace the pancreatization to this change.

In connection with the description of white hepatization, Virchow mentions another quite rare condition which he has seen several times in adults, every time in young girls. He says: This congenital alveolar catarrh is complicated by a condition which belongs to brown induration, but differs from the usual form in so far as no cardiac lesion, in fact no demonstrable impediment to the circulation outside of the lungs, is present, and, in spite of this, a considerable quantity of a brownish pigment accumulates within the lungs, chiefly in the cellular contents of the alveoli. The latter contain such a large number of catarrhal round cells that the tissue on account of this accumulation feels more dense and the entrance of air into the alveoli is rendered difficult. Most likely the alveolar catarrh is at the same time the cause of the impeded passage of the blood through the lungs.

Regarding the occurrence of the various pulmonary diseases after acquired syphilis, Virchow remarks, concerning the gummy nodules mentioned before as form 1, that quite a number of authors have described the finding of such nodules in the lungs, and although some of these descriptions give rise to some doubt, he is far from doubting the correctness of all the observations. According to his experience, he only wishes to recommend great care in giving an opinion. Caseous, yellow, and yellowish-white, principally encapsulated nodules are very frequent in the lungs, and present all grades of consistency, without there being any reason to take them, on this account or on account of their age, to be of syphilitic origin. The mere coincidence with other syphilitic products cannot be decisive here, for why cannot a syphilitic diseased person be attacked by tuberculosis of the lungs, and why not a phthisically affected individual by syphilis? "A great variety of nodular formations are found in the lungs. There are fibrous nodules, both white and pigmented, chiefly black; there are also dry, cheesy nodules, mostly white or yellowish-white in color, sometimes 'pepper and salt,' and these are in the highest degree suspicious. But peribronchitis and chronic pneumonia may give rise to similar formations even in cases in which there is no evidence of preexisting syphilis, so that it cannot be positively asserted that such nodules are actually gummatous. Even the coarse, nodular, extremely hard variety, which is occasionally found in constitutional syphilis, may develop without any signs of specific disease in stone-masons and individuals engaged in similar pursuits."

Nevertheless it is probable that the cases designated by Wagner as "pulmonary syphiloma" were true cases of syphilitic disease. Case XXV is quite typical; the pulmonary findings in this case were as follow:

Left pleura empty, lung clear, upper lobe of normal size, lower a little smaller, the former moderately emphysematous at the edges. In the upper lobe are a few nodules, mostly at a distance from the periphery, up to the size of a walnut, irregularly round and serrated, sharply defined, which are airless, homogeneous, greenish-gray or pale brownish-red, partly dotted black, slightly moist, and moderately firm. The remaining tissue of the upper lobe is anemic and moderately pigmented. The lower lobe firmer, heavy, and interspersed with numerous airless nodules, some as large as a goose-egg and similar to those in the upper lobe. The interior of most of them contains small, distended bronchi of cylindric shape, filled with grayish-yellow pus, the mucous membrane of which is dark red and much swollen. The tissue between these nodules is partly normal, partly almost or quite airless, blackish-gray, smooth, more firm, dry, especially where the nodules are found close together. The larger bronchi contain a small quantity of grayish-yellow mucus; the mucous membrane is moderately red and swollen. Bronchial glands slightly enlarged, black, and firm. Right lung adherent only over the posterior part of the upper lobe, otherwise the same as the left lung. The upper lobe in its upper half normal, in the lower interspersed with the same kind of nodules as the upper left; the remaining tissue of the same lung is moderately edematous and contains a moderate quantity of blood.

The middle lobe is anemic, but otherwise normal. The lower two-thirds of the lower lobe are in the same condition as the left lower lobe. The upper third contains air, is fairly vascular, and interspersed with some fresh hemorrhages up to the size of a bean. Bronchi and bronchial glands are the same as on the left side.

Wagner remarks, in connection with his casuistic description, that the nodules of pulmonary syphiloma are in the fully developed state are so characteristic that the fact of their having been almost entirely overlooked up to now can only be ascribed to the tendency to regard all caseous or dry tumors of the lungs as tubercles, and all softer ones as carcinomata.

Henop also gives a clinical and anatomic description of a case of nodular syphiloma of the lungs which in a very early stage of syphilis terminated in death. While the lungs were absolutely intact, nodes, some as large as a goose-egg, were found in both lungs. One of them occupied the greater part of the anterior surface of the right upper lobe; had a yellowish-white, slightly mottled gray appearance, on account of some pigment streaks; without vessels; resistant to the finger; and on the cut surface projecting above its surroundings. A layer, 2 mm. broad, of connective tissue which contained air separated it from the pleura. It deserves to be especially mentioned that a very instructive illustration accompanies the description.

Regarding the appearance of simple connective-tissue hyperplasia of the pulmonary tissue as a consequence of syphilis, Virchow maintains, with reference to the observations of Vidal and Dittrich, that undoubtedly there also exist simple irritative affections of the lungs of syphilitic origin which never become gummy. He observed them in the pulmonary parenchyma proper as well as in the finest bronchi and in the pleura, and he is inclined to regard certain fibrous forms of pleuritis, of pneumonia, and of peribronchitis as analogous with the chronic syphilitic inflammations of the liver, testicles, etc.

Probably Rollet was the first to demonstrate that pulmonary syphiloma appears not only in nodular masses of round, ellipsoid, or irregular shape of different size, sometimes as large as a fist, and either single or dispersed over larger parts of the lungs, but also forms diffuse infiltrations, which alone or in connection with nodules may extend over a pulmonary lobe.

In the same manner Schnitzler describes diffuse syphilitic disease of the lungs. In this affection the organ is generally of a firmer consistency, of heavier weight and more uniform surface. The infiltration extends over one or both lungs, or over only a part of them. The infiltrated parts are mostly entirely airless, grayish-red, grayish-yellow, smooth, homogeneous, with a scanty turbid juice. The bronchi are mostly of normal width and filled with much purulent mucous and little air.

Hiller describes exhaustively a case of gelatinous infiltration which affected the right lower lobe of the lung and was accompanied by pronounced syphilitic changes in the larynx, bronchi, liver, and bones.

The lower lobe, in its entire extent, showed a peculiar, grayish-white gelatinous infiltration of gummy elastic consistency, the parenchyma being absolutely airless. On pressure a turbid, gray, tough, almost colloid fluid could be squeezed out of the cut surface of the lung.

Pavlinoff also describes as a consequence of syphilis, besides interstitial inflammation and nodular peribronchial infiltrations of the left lung, changes in the upper and middle lobes of the right lung which he takes to be chronic parenchymatous (catarrhal) pneumonia. The cut surface was of a dark slate and grayish-red color, at the same time uneven from peribronchial nodules of different size which again were separated by isolated healthy or slightly infiltrated pulmonary parenchyma. In the right apex two bronchiectatic cavities of hazelnut-size were found; also at different places smaller saccular bronchiectases. The parenchyma of these lungs was intersected by broad streaks of glistening, firm connective tissue.

I myself have observed the following case, which may be included in the sphere of purely pneumonic diffuse consolidations, brought about by syphilis:

Mrs. F., forty-two years of age, was admitted to the hospital on March 7, 1896. She had formerly been a prostitute for many years, and, when twenty-three years old, was infected with syphilis. When twenty-four years of age she went through a puerperium which was complicated by abdominal inflammation. For a year she has been very short of breath when mounting stairs; for the past six months she has had a feeling of constriction about the chest, has suffered much from cough, which is very severe, so that sometimes her face turns almost blue. During the last six months she has lost 24 pounds and now weighs 62 kilos, about 124 pounds. Her face has a slightly livid appearance; the respiration is difficult. Nothing abnormal can be found in heart or lungs; the larynx is clear, the vocal cords are pure white, the tracheal cartilage is plainly visible.

The administration of potassium iodid caused so marked an improvement that the patient was discharged four weeks after her admittance, free of cough and dyspnea.

But after four days she returned, complaining of grave attacks of apnea. The attacks reappeared very frequently and could only be controlled by morphin injections. After the prolonged use of potassium iodid improvement took place. Although not quite free from dyspnea, she asked to be discharged on June 26, 1896.

Four weeks later, on July 26th, she was again brought to the hospital, suffering from intense dyspnea, stertorous breathing, and cyanosis of the face. While so far the lungs had appeared to be normal and the symptoms had been ascribed solely to a syphilitic stenosis of the large air-passages, dulness and fine râles were not discovered on the right side of the thorax, from the middle of the scapula anteriorly downward. Fever had never been present; now the temperature in the axillary space was 38.3° C. (101° F.). The urine contained a little albumin.

The dyspnea continued practically unchanged during the following days. Subcutaneous injections of morphin and potassium iodid failed to give relief. Auscultation of the trachea over the jugulum sterni showed only very slow respiration which could hardly be called bronchial.

After twenty-four hours the albumin had disappeared from the urine. The fever continued, and, strange to say, was higher in the morning than in the evening; it was, on July 30th, A. M., 39° C. (102.2° F.); P. M., 36.6° C. (97.9° F.); on July 31st, A. M., 39.3° C. (102.7° F.); P. M., 36.8° C. (98.2° F.).

On August 1st death occurred with continued dyspnea in a state of somnolence.

Only the condition of the thoracic organs at the autopsy need to be mentioned here, as the other organs did not show any abnormality.

The pericardium contained a small quantity of clear serous fluid, and was very fatty; the muscular structure of the heart was flabby and pale brown; all valves were intact.

Both pleural cavities contained a small quantity of clear serous fluid. The upper and lower lobes of the right lung were well filled with air and blood; the middle lobe, however, was almost airless, of cloudy tough consistency, the cut surface pale gray, not granular.

On the left side of the trachea, immediately over the bifurcation, a prominence is present which leaves only a narrow, crescent-shaped slit within the tube. The entrance of the left bronchus is obstructed by the prominence, which arches over the mouth of the tube. This prominence corresponds to a fluctuating tumor which is located entirely outside of the tracheal wall, so that the tracheal cartilages and mucous membrane, which bulge inward, are intact except a small, lentil-sized spot on top of the tracheal prominence which corresponds with the mass filling the tumor: namely, coagulated blood—as is shown by a bisection of the tumor. The tumor itself is formed by an almost globe-like dilatation of the aorta as large as a plum. This dilatation communicates with the lumen of the arch of the aorta by a circular opening 1 cm. in diameter. The inner surfaces of both the ascending aorta and the arch show numerous contiguous eminences of yellowish-white color, some of which feel as hard as bones.

In addition to the syphilitic infection which was known to exist, only the following secondary conditions were to be observed in this case: The atheroma of the aorta, which was of very high grade considering the age of forty-two years, with an aneurysmal dilatation depending on the same which caused a compression of the larynx, and furthermore a pneumonic consolidation of the middle lobe. If I consider syphilis to be the cause, I do so not only on account of the communications referred to, according to which the middle or lower lobes were diseased in syphilis, and the clinical observations, to be explained later on, according to which the middle lobe is the seat of predilection of syphilitic pneumonia, but principally on account of the microscopic examination of the case in question.

All the alveoli were filled with cellular material. Only a few contained swollen alveolar epithelia exclusively; not much more numerous were such alveoli, in which a smaller number of white blood-corpuscles were embedded in fibrinous masses, while the alveolar capillaries were distended with blood. But most of the alveoli appeared to be well filled with small round cells interspersed with red blood-corpuscles. A much larger quantity of the latter was present in the interalveolar and interlobular tissue.

This, however, does not prove that this pneumonic inflammation is entitled to a special classification, the only justification for which would be a considerable increase in the thickness of the walls of the smaller vessels. Exactly as described in the definition of white syphilitic pneumonia of the newborn represented on Plate 7, figure 8, there exists in this instance an increase of the elements which form the muscularis and the adventitia, and here also the adventitia is principally affected.

Consequently, according to the above communications of Rollet, Schnitzler, Hiller, Pavlinoff, and according to my own description, the occurrence of diffuse pneumonic disease secondary to syphilis may be considered to be positively proved.

Furthermore, the question should also be discussed whether it be possible that gummy inflammation, or diffuse inflammation of the connective tissue, or parenchymatous inflammation can cause a process of disintegration in the lungs with the formation of cavities like those caused by tuberculosis.

Based on a very careful analysis of the previous reports in reference to syphilitic phthisis, Hiller criticizes the want of care so far displayed in the diagnosis of this affection. The entire literature of syphilis of the lungs shows that, almost without exception, in cases of pulmonary syphilis that have come to autopsy, changes different from those found in phthisis have been demonstrated; that is, the authors in these cases evidently had to deal with pulmonary syphilis, while, on the other hand, no autopsy has ever been made in a case described as syphilitic phthisis, because cure or improvement was effected by proper antisyphilitic measures.

This standpoint of an objective criticism is certainly well founded; especially in view of the fact that the influence of syphilis upon the origin of disintegration processes in the lungs has been greatly overestimated. But does not the question of syphilitic phthisis deserve to be investigated from another point of view?

We should not forget that the word phthisis originally designated only a clinical conception. Based upon this meaning of the word, Bayle has already distinguished six different kinds of phthisis according to the originating causes. In this sense his predecessors and his contemporaries referred to a syphilitic phthisis of the lungs.

To-day, when phthisis of the lungs and the tubercle bacillus are justly considered to be identical, the designation "syphilitic phthisis" may be discarded.

Therefore we can only refer to syphilitic inflammation of the lungs, or, as Hiller advises, to syphilitic infiltration; but we would considerably lessen our own knowledge of the same if we neglected to consider the experience which up to now has been gathered under the title "syphilitic phthisis," especially if the data obtained are discussed with a view of clearing up the differences between changes in the pulmonary tissue caused by syphilis and tuberculosis of the lungs.

The view of Hertz that in the case described by him the pulmonary changes and partial disintegration of the tissue are to be ascribed to syphilis is probably well founded.

The case was that of a woman, thirty-four years of age, who had been infected with syphilis two years previously. Clinically she offered a number of symptoms which pointed to the presence of an aneurysm of the ascending aorta; cerebral embolism; and pneumonic infiltration of the entire lower, and part of the upper lobe of the left lung, with commencing infiltration in the lower part of the right lung. Autopsy proved the correctness of the diagnosis. The upper half and the anterior edge of the left upper lobe were completely filled with air. Further down and posteriorly the parenchyma was interspersed with numerous nodules varying between the size of a millet-seed and that of a small pea. They were irregularly defined, of gray and white and even blackish color, and between them parenchyma was found which mostly still contained air. The base of the upper lobe, however, was occupied by an almost gray appearing consolidation, uniformly diffuse, or appearing in larger spots, which was interrupted by delicate, reticular, golden tracings (fatty degenerated epithelium), and by yellow, caseous, disintegrating softening areas. Intersecting those uniformly diffuse consolidations were streaks of connective tissue which divided the parenchyma into several sections; a large number of these hard, pea-sized nodules were found here also. Almost the entire lower lobe showed a focal infiltration, gray on the cut surface, with yellow central softening, which was also intersected by firm, dense streaks of connective tissue. This more uniform, pneumonic infiltration was amply interspersed with coarse, grayish-white nodules, part of which contained a yellow tough mass in the center which could be removed by pressure. The bronchi in the infiltrated parts of the upper lobe and in the entire lower lobe were considerably dilated, their mucous membrane was very much congested and filled with a tough puriform mass which could be drawn out into long strings.

The right lung had a very large volume. Well-marked vicarious alveolar emphysema existed in the upper lobe with little edema and little blood contents. The greater part of the middle and lower lobe showed the same conditions. Only at the lower edge of the middle, and at the base of the lower lobe numerous foci of gray consolidations were formed with caseous center which were inclosed in a uniformly grayish-red, more flabby infiltration. The pleura over the infiltrated parts was covered with a delicate layer of exudation, under which the pulmonary surface showed an unequal, rough condition similar to the above-mentioned nodules; the pleural tissue itself was interspersed with the same oval, white nodules. The bronchi in the infiltrated connective tissue appeared dilated, much congested, and filled with tough puriform masses; in the air-containing parenchyma, on the other hand, the bronchi were normal and contained only a frothy secretion.

According to Hertz's opinion, the above-described gray and grayish-white areas, partly interspersed with black pigment, proved to be peribronchitic proliferations which originated in the bronchial walls and were composed of formations which increased in age in proportion to the distance from these walls. The uniform infiltrations, without nodules, represent areas of lobular pneumonia in the interstitial connective tissue and in the neighboring alveoli which reveal a beginning phthisis by the presence of partial disintegration. Besides the peribronchitic nodules and the lobular pneumonic infiltrations, a very considerable purulent bronchitis was found with partial dilation of the bronchial branches. The fact that the bronchi, in those parts which were free from nodules and lobular infiltrations, appeared healthy throughout, and that the intensity of the affection in the peribronchitic nodules and lobular infiltrations was in keeping with that of the parenchyma, does not permit us to trace these nodules and infiltration to a primary bronchitis. Accordingly, it would seem that the process originated in the connective tissue of the bronchial walls and in the interlobular connective tissue, and that the catarrhal process did not set in until later, possibly as the result of circulatory disturbances in the mucous membrane of the bronchi and in the pulmonary lobes. But there is a possibility that these lobular changes are effected by an exudative process immediately depending upon syphilis, and synchronous with the hyperplasia of the connective tissue; by a so-called albuminous infiltration or saturation of the epithelial cells of the pulmonary alveoli with a gelatinous nutritive material which soon leads to retrogressive metamorphosis and cell-necrosis.

In the case described by von Cube it may also be assumed without any doubt that the expectoration of pieces of pulmonary connective tissue was the symptom of a destructive process developing on a syphilitic base. This case terminated favorably after antisyphilitic treatment. There was persistent high fever with distressing cough, and the expectorated material on microscopic examination was found to contain detritus, mucus and pus-corpuscles, fibrin, fibrous tissue, cells containing pigment granules, and in some places large masses of elastic fibers which were mostly arranged in a certain order.

The sputa in the case described by Güntz contained only fine granular detritus, in places, with many shrunken nuclei and granules, remnants of finely striated stroma, a few spindle cells, here and there fine masses of fibers, crossing each other in all directions; but no blood-corpuscles, pulmonary tissue, blood-vessels, were anywhere to be found.

Finally, the combination of syphilis and tuberculosis is to be taken into consideration. Although Schnitzler was the first to report a case of tuberculosis and syphilis of the respiratory organs, it is chiefly Potain who discussed the combination of these two processes with particular reference to the tubercle bacillus which is characteristic of tuberculosis. He treats the subject from two points of view: (1) Syphilis occurring in an individual known to be tuberculous; (2) tuberculosis occurring in a syphilitic person.

1. According to Potain, tuberculosis is aggravated if complicated

by syphilis, because the symptoms of the latter are added to those of the former, and injudicious antisyphilitic treatment tends still more to debilitate the organism.

2. If tuberculosis attacks a syphilitic person, it is generally during a later stage of syphilis. The latter seems to favor the growth of the tubercle bacillus. He declares syphilis to be a predisposing cause of tuberculosis, and in support of this view cites a report of Sokolowsky.

The pathologic alterations produced when an already existing tuberculosis, with infiltration and cavity-formation, becomes complicated by a severe syphilitic infection have been described by Rindfleisch. The pulmonary apex contained a cavity as large as a pea which formed the center of a nodular infiltrate, surrounded and crossed by thickened bands. Some of these nodules were as large as hemp-seeds and represented true syphilitic gummata; while other, smaller ones, were genuine miliary tubercles. The latter were undergoing fibrous metamorphosis. Even the giant-cells were breaking up, in places, to form fibroblasts. Masses of miliary tubercles were surrounded by thickened connective tissue which was continuous with the thickened bands in the septa between the lobules. Numerous tubercle bacilli were found in the cavity. Rindfleisch believes that the development of a tuberculous granuloma, especially a miliary tubercle, is modified by the appearance of syphilis and becomes encapsulated, like a fully developed structure, in a mass of luetic fibroid tissue; in other words, syphilis is capable of exerting a curative action on tuberculosis.

SYMPTOMATOLOGY AND DIAGNOSIS.

After what has been said in regard to the pathogenesis, it is unnecessary, in the description of the symptomatology, to speak of any changes other than those produced by acquired syphilis. Even if white hepatization in the newborn were easy to recognize and to distinguish from simple atelectasis, the number of living infants delivered at term is so small that the question of diagnosis need not be considered.

In view of the impossibility of making an accurate analysis of the symptoms, any attempt to distinguish clinically between the various pathologic processes would be impracticable. It is impossible during life to distinguish individual gummatous nodules in the lungs, even when they are as large as in the case described by Wagner and Henop, from simple fibrous thickening due to peribronchitis, a condition that unquestionably occurs, especially if the pulmonary tissue takes part in the chronic inflammatory changes, or the gummatous nodules are associated with simple fibrous hyperplastic processes, and, as frequently happens, according to Fournier, are quite extensive and often occupy an entire pulmonary lobe.

To make a sharp clinical distinction it is necessary to demonstrate the occurrence of lobar affections consecutive to pneumonic inflammations, the existence of which has been established on pathologic grounds by Rollet, Schnitzler, Hiller, Pavlinoff, and myself.

It is hardly possible to clinically consider more than two kinds of syphilitic disease of the lungs, namely: (1) Syphilitic lobar pneumonia; (2) chronic, fibrous, hyperplastic consolidations, which mostly originate in the bronchi and in the peribronchial connective tissue, sometimes associated with the presence of gummy nodules.

At the same time, it must not be overlooked that even the presence of all the subjective and objective signs in the lungs does not in itself suffice to establish the diagnosis, and that every additional datum, derived either from the history or from a general examination of the patient (?), must be carefully utilized.

It will be necessary to observe for the clinical demonstration of lobar-pneumonic affections that, according to Grandidier's report, the right middle pulmonary lobe is most frequently the seat of this affection. He observed this condition 27 times in 30 cases. Then a conspicuous dulness exists, corresponding to the interscapular region as far as the middle of the base of the right scapula. This dulness is most intense in the neighborhood of the hilum of the lung, gradually disappearing upward, downward, and laterally. Apex and base of the lung are always intact in the stage of interstitial pneumonia, but later on physical changes set in here as well. At the same time a dulness is found anteriorly in the second and third right intercostal spaces near the sternum; rarely the left lung is affected. Respiratory sounds are absent in the dull areas, in which bronchial respiration can sometimes be noticed. While feeble respiratory sounds can be observed over the lower corner of the right scapula, the respiratory sounds over the lower lobe of the right lung are generally clear and normal.

Grandidier declares this infiltration, which is generally limited to the middle lobe of the right lung, to be an important point in the differential diagnosis from infiltration of the lungs in tuberculosis and in caseous pneumonia. He believes that this localization in itself is so significant that, when it is present, other symptoms can be dispensed with in order to determine the syphilitic nature of the pulmonary disease.

Schnitzler also has mentioned convincing clinical instances for this peculiar preference of syphilis for the middle and lower lobes of the lungs. His first case of this kind was that of a woman, thirty-two years of age, who six years after infection was attacked by gummata and ulceration of the larynx, and at the same time suffered from a pneumonia of the middle pulmonary lobe, which was completely cured by the administration of large doses of potassium iodid. Two years later the patient was again treated for ozena and infiltration of both lower lobes and of the middle lobe. The pulmonary affection was again cured by the use of iodid of iron.

The second case was that of a man, twenty-five years of age, who, five years after infection, was treated for ulcers in the soft palate, gummata on the epiglottis, ulcers over the right arytenoid cartilage as well as on the vocal cords, and in whom "at the right base posteriorly bronchial respiration was present, furthermore over the entire lung rough indistinct breathing with coarse and fine crepitant râles." After the use of potassium iodid—at first 2 gm. (30 grains), later 5 gm. (75 grains) a day—all symptoms of disease disappeared; after several weeks' treatment not a trace of syphilitic infiltration could be detected by means of the most minute physical examination.

The third case was that of a female laborer, who complained of hoarseness, cough, and severe dyspnea. The percussion notes were slightly shortened over both apices posteriorly as well as anteriorly, the respiratory sounds were rough and indistinct, covered by a loud laryngo-tracheal stenotic sound; extensive dulness could be demonstrated over the inferior angle of the left scapula, where bronchial respiration and sometimes crepitant râles could be heard. The diagnosis "tuberculosis of the lungs," made on the strength of these findings, was not discarded until the laryngoscopic examination showed ulceration of the epiglottis and of the vocal cords as well as a deep ulcer with ragged edges and floor covered with a purulent exudate over the left arytenoid cartilage, and it was accordingly ascertained that syphilitic infection had taken place six years before. Severe choking spells led to the suspicion of the presence of laryngeal stenosis. After five weeks' treatment (inunction cure) the larynx was almost normal. The ulcers on the vocal cords, on the arytenoid cartilage, and on the epiglottis were cured and left only scarcely perceptible traces. The voice was loud and strong, although still a little hoarse. The infiltration of the lungs had disappeared except a slight dulness in the apices; the cough was gone, and the dyspnea, the removal of which by tracheotomy had already been decided upon, existed only in the memory of the patient.

Quite in equal manner Pankritius lays stress upon the seat in the center of the lungs of syphilitic inflammation of the lungs. According to his statement, there exist during the first stage, in the interscapular region, *i. e.*, at the base of the scapula, corresponding to the hilum of the lungs, a dull percussion sound, weak crepitant râles, "especially decreased expiration"; in the second and third intercostal spaces anteriorly a full tympanitic note, often increased inspiration and decreased expiration, or weakened breath sounds. During the second stage dulness is present in the second and third intercostal spaces anteriorly. During the third stage the intense dulness at the base of the scapula has extensively increased; inspiration has become stronger, is often bronchial; expiration is abolished. Dull tympanitic notes are elicited in the second and third intercostal spaces anteriorly, "at the same time a depression of the affected intercostal space is observed; the latter on deep inspiration bulge and quickly collapse again. The excursions of the thorax appear somewhat restricted; the thorax decreases in circumference."

Pankritius explains the diversity in the results of physical examination by the course of the interstitial pneumonia which occurs primarily in the locality referred to [the middle and lower lobes.—Ed.], although, it is true, the parenchyma may also become involved. "If, however, a secondary parenchymatous syphilitic pneumonia becomes temporarily arrested, the specific process returns to its original seat, the connective tissue; but sooner or later, under the influence of some new irritation, the process resumes its activity in the parenchyma, so that intervals of apparent recovery alternate with periods when the hyperplasia of the connective tissue and the participation of the parenchyma are more marked."

Valuable as these clinical evidences of the characteristic localization of pulmonary syphilis may be, the diagnosis cannot very well be based on this fact alone, Grandidier to the contrary notwithstanding. When it is recalled that repeated attacks of pneumonia may leave residuary changes in the middle lobe and in portions of the lower lobe; that the physical signs elicited in bronchiectasis may be quite similar to the signs described by Grandidier as characteristic [of pulmonary syphilis]; and that tuberculous infiltrations may involve a portion of the lower lobe in addition to the apex, it is evident that the greatest caution is necessary and that every available diagnostic aid must be utilized.

If the disease, instead of presenting a lobular distribution, assumes the form of chronic hyperplasia of the peribronchial connective tissue resulting in partial consolidation of irregular areas in the lungs, it is even more difficult to arrive at an accurate diagnosis. Identical subjective symptoms and objective findings occur in other pulmonary affections, especially tuberculosis. First of all, the previous occurrence of syphilitic infection must be definitely established. For this reason it is impossible to accept without reserve the cases of Langerhans and Schiffmacher which presented the clinical picture of tuberculosis while the history contained no positive evidence of syphilitic infection, although mercurial treatment by inunction proved successful. The history, the symptoms, and the diagnosis must be mutually confirmatory, if the treatment is to rest on a secure foundation.

The infection may have taken place a long time before. According to Fournier, pulmonary syphilis sets in during the tertiary period, most frequently in a late stage of the same. Five, ten, fifteen years, sometimes even a longer time, may have elapsed since infection.

Lancereaux describes a rare case of hereditary syphilis of the lungs in middle age. The patient was descended from notoriously syphilitic parents, and in her earliest childhood showed manifold symptoms of lues. In her fortieth year she was attacked by hemoptysis, which after several months, with the usual symptoms of phthisis, terminated in death. At the autopsy the left lung appeared fairly normal. The middle and lower lobes of the right lung were consolidated, and traversed by broad fibrous bands; the consolidated tissue contained several hard, sharply defined nodules of different sizes; also several hard, sharply defined, smooth-edged cavities, which were entirely different from the usual tubercular nodules, and from the cavities that occur in tuberculosis. Furthermore, characteristic syphilitic changes were found in the iris and in the tibia.

In regard to the individual symptoms, cough, dyspnea, hemoptysis, sputum, and fever, it must be distinctly emphasized that they present nothing characteristic.

Cough is rarely absent, but it is not always proportionate to the severity of the disease. According to Schnitzler's experience, the severity of the cough, in pulmonary syphilis as well as in pulmonary phthisis, depends on the degree to which the larynx, the trachea, and the bronchi are involved; while true parenchymatous diseases of the lungs give rise to cough only when the peribronchial lymph-glands or the pleura are implicated.

Dyspnea is one of the most important functional symptoms. One of its chief peculiarities is that it cannot very well be explained from the objective findings. There is a disproportion between the dyspnea and the results of auscultation and percussion. It has been ascribed to a swelling of the bronchial glands or to compression of one of the larger bronchi by a gumma. More correctly, it may be explained by a flattening of the alveoli and a degeneration of the alveolar epithelium (Mauriac).

The sputa do not show anything characteristic. The presence of tissue particles has been previously mentioned (page 652).

Hemoptysis is rarer than in tuberculosis and usually smaller in quantity, but exceptions will occur. A patient of Lancereux expectorated more than 1 liter of blood in twenty-four hours; a patient of Carlier, 2 glassfuls in a single hemorrhage.

Fever may be absent entirely, or reach a high degree in other cases. The temperature cannot be utilized at all as an aid in diagnosis.

Auscultation and percussion, except over the above-mentioned region, merely reveal the presence of foci of consolidation of variable extent.

The presence of the remains of syphilis in other organs is of the greatest importance, and should always be looked for. In almost all the recorded cases of pulmonary syphilis, in which infection was positively known to have taken place, signs of the disease were found in some one of the organs or systems known to be the favorite seats of syphilitic changes; in the pharynx and larynx, in the trachea and its branches, in the osseous system, in the liver, and in other organs. Of course, if the pulmonary disease develops from ten to fifteen years after infection, the concomitant syphilitic manifestations may be confined to the vascular system. If the latter is severely and extensively involved, or an aneurysm secondary to atheromatous changes in the vessel-wall exists, especially in middle-aged persons, such findings constitute a valuable auxiliary for the recognition of the nature of the pulmonary affection, provided, of course, that syphilitic infection has taken place.

TREATMENT.

It is universally admitted that as soon as the syphilitic origin of a pulmonary affection has been recognized, no time should be lost in beginning antisyphilitic treatment. Mercury is the drug that best meets the indications. Its superiority to potassium iodid in large doses is attested by the available literature on syphilitic pneumonia and confirmed by the judgment of the majority of authors. Judging from my own experience, inunctions with mercurial ointment constitute the best method of administering the drug. As I consider this procedure absolutely innocuous if it is carried out under the proper medical supervision and with the observance of the necessary hygienic precautions, I do not hesitate to say that I follow the old custom of ordering the inunctions to be kept up for six weeks. During the first three weeks I prescribe 2 grams (30 grains) to be rubbed in every evening for six days in the week. During the last three weeks, in the absence of any disturbing consequences, especially severe salivation, the dose is increased to 3 grams (45 grains). The inunctions are practised on the leg, the thigh, the forearm, the arm, the abdomen, and the chest—a different part being selected each evening in the order named. Woolen underwear of a thickness to suit the season should be worn. On the seventh day—*i. e.*, after each series of six inunctions—the patient takes a warm bath. A mouthwash consisting of a 3% solution of potassium chlorate, or a weak alum solution, must be used from the beginning of the treatment.

Less agreement has been reached in regard to the questions: (1) What shall be done in those cases in which a syphilitic infection is not admitted or actually, according to the knowledge of the patient, has not taken place, in spite of several symptoms being present in association with disease of the lungs, which make a previous syphilitic infection probable? and (2) What is to be done in cases in which a syphilitic infection is known to have taken place previously, but where the affection of the lungs proves to be tuberculous beyond doubt, as tubercle bacilli are present in the sputa?

In answer to these two questions we are justified by experience in saying that, as a rule, antisyphilitic treatment is permissible; and if Potain's experience goes for anything, even when a syphilitic subject becomes secondarily infected with tuberculosis. He says: "Even if Koch's bacillus is found in the sputum, the syphilitic process must be attacked because it aggravates the symptoms. It increases the already existing cachexia, whereas antiluetic treatment exerts a favorable influence on the syphilitic, as well as on the tuberculous pulmonary manifestations. Hence a syphilitic suffering from tuberculosis should not be treated for his tuberculosis alone, to the neglect of the syphilitic symptoms. Of the two diseases, it is the latter that can be more rapidly modified by treatment, and it must be prevented from aggravating the tuberculous condition. By curing the syphilis the greatest boon is conferred on a patient who has Koch's bacillus in his lungs. Antisyphilitic treatment should therefore be begun at once, even if the syphilitic manifestations are quite insignificant."

However, it must be mentioned that Hiller is of a different opinion. He maintains that every antisyphilitic medication is more or less harmful and that phthisical patients especially are generally not well able to bear it. In 44 patients medication could be carried through to the end (40 injections) in only half the cases. In fact, in well-advanced cases of phthisis the effects of the sublimate were absolutely deleterious, especially in such patients as were continually bedridden and feverish; in these cases it promoted debility, and in several instances it was materially conducive to the fatal termination of the disease.

These objections do not appear to me to be quite valid. It is true I can only offer one observation in rebuttal, but it ought to be sufficient materially to weaken these weighty objections to the antisyphilitic treatment of notoriously tuberculous patients. My observation concerns a man, twenty-four years of age, whom I treated for three years for severe infiltration of the apices of the lungs which at the beginning was accompanied by fever. Numerous tubercle bacilli were found in the sputa. Soon after the onset of the affection a pleuritis set in which was demonstrated by aspiration to be serous, and the effusion of which filled almost the entire left side of the thorax. After several weeks it disappeared almost entirely, but dulness over the apices and catarrhal manifestations continued and have not quite disappeared yet. But in spite of this the young man was able, after considerable improvement of the general state of his health had been brought about by sending him to health resorts, to follow his easy occupation. Then, about a year ago he became infected. After roseola and ulceration of the pharynx had set in, a thorough inunction treatment was carried through with careful consideration of other hygienic conditions, and all the symptoms of syphilis were removed. He lost only 2 kilos during the treatment, and made this loss good in a short time. At present, six months after the treatment, he is rather stronger than before infection took place, and has even engaged in a profession requiring more exertion.

PNEUMONOKONIOSIS.

PATHOGENESIS.

The view that inhaled dust enters into the pulmonary tissue has been advanced by famous authors since Pearson's publication in 1813. But the conclusive power of the observations reported by him was insufficient, in view of Henle's doubts and Virchow's investigations. The former declared it to be impossible that the black pigment in the lungs should consist of inhaled soot and coal-dust particles, because then it could not be explained how they would get into the bronchial glands, and even into the lymph-passages; and Virchow, by his report of the origin of pathologic pigments, made the view untenable for the time being. He based his investigation of their presence in the pulmonary tissue on the changes that occur in brown induration in consequence of cardiac lesions, and believed himself justified in regarding the finding of black pigment granules in the epithelial cells of the lungs as a gradual change of yellow and brown pigment granules which are found in other cells.

A thorough description here of the observations which were made until Virchow's report appeared is the less required, as they are exhaustively reported in the easily accessible treatises, published by von Fenker, Hirt, Merkel, and dealing with those scientific works which defend the penetration of coal-dust. Neither would it be in keeping with the object of this work to present the extremely abundant literature of this subject, and it may therefore be permitted to start from the investigation of Traube, who, more convincing than all former ones, has brought about a complete change of opinion.

In Traube's case the sputum consisted of a small number of gray, translucent, punctate balls which contained, besides numerous mucus-corpuscles, a fair number of larger cells, filled with black molecules, and an abundance of black particles which were not inclosed in cells. These black bodies showed a sharply defined, angular, but at the same time irregular shape throughout. This fact and the considerable size of many of these forms suggested the thought that it was not a question of black pigment, but possibly of inhaled coal-dust. The patient, upon inquiry, stated that for the last twelve years he had been engaged in loading and unloading charcoal. At the autopsy the cut surface of both lungs, except in a few places, was of a uniformly black color, smooth throughout, glistening, and as soft to the touch as normal pulmonary parenchyma. On pressure a frothy serous fluid, at some places very copious, exuded everywhere. It was of a black color and stained the finger like inferior black water-color. No bands of newly formed connective tissue or indurations were anywhere to be seen, which later was confirmed by the microscope.

The examination of the black fluid discharged from the cut surface of the lungs showed: (*a*) The same manifold formations of black and red particles which, during life, had been found in the sputum; (*b*) black globules of different sizes, many of them five to six times larger than common pulmonary epithelia, consisting of a dense aggregation of the described black particles; (*c*) single cells, resembling pulmonary epithelia in form and size, in which, besides the nucleus, smaller and larger black angular bodies were present which sometimes were as long as the largest diameter of a cell.

The microscopic examination of the smallest particles of common charcoal showed an absolute conformity with the bodies found in the sputum and in the lungs.

From his investigations Traube concludes: (1) That bodies, usually foreign to the organism, may have penetrated into the cells without causing their disintegration or even any noticeable changes; (2) that the penetration of the sharp bodies has occurred because particles which are driven by the inspiratory air-current enter the alveoli with a certain velocity and are very apt to perforate the opposing cellular walls; (3) as many people continually live in an atmosphere loaded with coal-dust without showing any symptoms of bronchial or pulmonary disease, another circumstance must exist to cause the aggregation of fine particles in the alveoli of the lungs. In this case the fact that the patient had been suffering from bronchial catarrh before he worked in the coal-yard led to the inference that possibly a disturbance of the ciliary motion represented the second condition.

Maurice declares likewise that the aggregation of black pigment in the lungs consists of coal, and is caused by the direct entrance of coal-dust, not by a secretion from the blood. After reviewing the history and the autopsy of a coal-laborer whose lungs were interspersed with numerous hard, miliary nodules, he arrives at the remarkable result that while the entrance of coal-dust into the lungs is borne out by the autopsy in all coal-miners, the quantity is rarely as abundant as in the case reported. The reason for this is found in the presence of miliary tuberculosis, which decreases the elasticity of the pulmonary vesicles in the neighborhood of the nodules, and thereby, first, interferes with the renewal of the air, and favors the depositing of the inhaled coal-dust; and, secondly, inhibits the expulsion of the deposited coal. For this reason the densest deposits of coal were found in the neighborhood of the tubercles.

Furthermore it was possible to demonstrate the entrance of coal-dust into the pulmonary parenchyma, its penetration into the pulmonary alveoli, and its presence in the bronchial glands in the case reported by Leuthold from Traube's clinic. But here cavities in the upper lobe of the lungs and miliary nodules were also found. An etiologic reference of the aggregated coal particles to the pathologic changes had to be excluded.

Now, Virchow also, in connection with the unsuccessful experiment of Koschlakoff to demonstrate by chemical means differences between inhaled coal-dust and locally originated pigments, and with

Traube's observation, expressed the opinion that larger fragments of vegetable coal might penetrate into the alveoli and "accumulate there to the utmost." The absolutely characteristic forms of this charcoal preclude any error; from that moment on he had no more doubts that there existed a real anthracosis of the lungs. Since then he had several times the opportunity of seeing similar, although not quite as pronounced cases; and after these experiences he declared it to be not only very probable that all these sharp, angular, large bodies, which he formerly took for black crystals, are fragments of charred wood- or vegetable cells, but he also inclined to ascribe some of the fine, granular, black pulmonary pigment to the soot and smoke of burning black bodies, after he had repeatedly observed the change of fine coal-dust into catarrhal cells. This, however, does not prevent him from excepting those pigments in which yellow and brown granules gradually turn into black ones, as is the case in extravasation into the lungs, sometimes, it is true, besides in connection with anthracosis. Even now he pronounces brown induration to be the most convincing proof of this process. Consequently he makes a distinction between anthracosis proper and pigmentation of the lungs.

A valuable furtherance of the much discussed question was reached by observation of cases in which the deposit of other kinds of dust in the pulmonary tissue had recurred and the presumption was precluded that they might have arisen in the organism. Zenker had a chance to examine the lungs of a working-girl who in her occupation with red paint had for about seven years inhaled dust of oxid of iron. Both lungs were colored an intense brick-red. The cut surface appeared as a uniform, fine cellular, red meshwork. The bronchi also contained some of the coloring mass which was discharged from the cut surface as a turbid brick-red fluid. In all lobes of both lungs were scattered numerous fibrous nodes, from the size of a pinhead to that of a pea, some even larger, of yellowish-gray color on the cut surface, which were interspersed with smaller and larger brick-red spots and partly showed black spots. Furthermore, all the pulmonary lobes, except the middle one, contained irregular sinuate cavities. Brick-red spots appeared in the finest bronchi, and, according to the microscopic examination, were formed by dense granular deposits in the deeper layer of the bronchial wall, while the superficial layer was not stained. The bronchial glands at the bases of both lungs were of normal size, mostly black near the center and intensely red on the surface. "The most abundant deposits were found in the alveolar epithelia—the interalveolar septa were free—furthermore in the lobular and infundibular septa, in the sheaths of the finer bronchi, in the subpleural connective tissue, and in the lower layer of the pleura itself. Therefore the dust particles after their penetration of the alveolar wall proceed in these connective-tissue parts even as far as into the pleura."

Zenker is inclined to consider the described nodes and cavities to be a consequence of the damage to the tissue by the inhaled oxid of iron dust.

His designation "pneumonokoniosis" for the pulmonary changes brought about by different kinds of dust has probably been universally accepted.

In addition to coal-dust (anthracosis) and that of iron-dust (siderosis), another kind of dust is to be considered, which, as a fact, represents a mixture of different kinds of inorganic substances, but, on account of the prevailing stone-dust, is called *chalicosis pulmonum*. According to Merkel, this kind of pneumonokoniosis is one of the first dust-inhaling diseases which was carefully observed and reported. As early as 1703 Ramazzini reported the autopsy of such a case, and the first positive proof of deposited dust-molecules was produced in 1860 by Peacock. He microscopically demonstrated the quartz particles in the ashes which were extracted by muriatic acid. Kussmaul demonstrated in the lungs of a stone-cutter, which in a characteristic manner showed the symptoms of chronic bronchitis with melanotic consolidation of the tissue, three times as large a quantity of sand as is usually contained in the lungs of adults who do not "work in stone."

Lately Woskressencky has demonstrated that sand as well as coal particles are present in the lungs of all men. It gets in from the air into the lungs; its quantity depends upon the occupation and is in proportion to the age. The lungs always contain less silicic acid than the bronchial glands.

In a miller, sixty-five years of age, the proportion of silicic acid in the lungs to ashes in the glands was as 33.7% to 55.6%; in a soldier, twenty-five years of age, it was as 7.3% to 34.4%.

It is obviously beyond the province of this treatise to give a statement of those professions which may cause an affection of the lungs by inhalation of different kinds of dust; the text-books of trade-hygiene, of which those of Hirt and Merkel deserve to be mentioned before all, give a very explicit report of the same. We shall merely consider the leading standpoints for judging of the pathogenesis of the affection. Therefore it shall only be mentioned that, before all, the observations reported in 1866 by Greenhow of pulmonary changes in a stone-cutter and in a potter belong here, also the cases in which the dust of soapstone is inhaled in working up soapstone into gas-burners, of which Merkel, Thoral, and others have reported.

On account of its conspicuous importance for the origin of acute inflammatory changes of the lungs, the inhalation of Thomas phosphate deserves exhaustive consideration.

Ehrhardt, in his report, which is fundamental for this subject, justly opines that the considerable amount of dust which is caused by the manufacture of phosphorus-meal out of Thomas slags, not only by its quantity but also and principally by its chemical composition, becomes an absolutely harmful agent for the laborers who work in the dusty atmosphere. As physician in one of the extensive establishments for the manufacture of this phosphorus-meal, he became convinced that no laborer had worked in these factories without more or less harm to his respiratory organs. As a consequence mostly severe pneumonias appeared, the frequency of which—during the first five months of the works being operated, 48%; during the following year, 1888, 25.5%; and in 1889, 21.5% of all their men—proved in itself the detrimental effect of the inhaled dust. Still more convincing is the fact that absolutely healthy laborers who commenced to work in these factories were attacked by this inflammation of the lungs after scarcely two weeks, and again others who had overcome such an inhalation pneumonia, shortly after their re-entrance into this dust atmosphere were again attacked by the disease, sometimes as often as four times in succession.

The course of the disease is a very unfavorable one; 6 died out of 20 who were affected by pneumonia.

If, however, Ehrhardt considers the purely chemical element to be of influence in the origin of the disease, and concludes therefrom that the pathogenesis of croupous pneumonia from a purely bacteriologic point of view cannot be sustained, I cannot agree with him. The absence of Diplococcus pneumoniæ does not prove that the origin of the disease was purely chemical. In fact, after investigating the clinical report of the twenty cases described, and in consideration of the results of the four autopsies, I would have, not exactly excluded, but surely not presumed the finding of the Fraenkel-Weichselbaum diplococcus, because, according to their clinical and anatomic course, these cases are not to be considered to be croupous pneumonia, but atypical pneumonia, as described on page 579.

During his observations in the Thomas phosphate mill, situated at Ruhrort am Rhein, Greifenhagen found that the workmen employed in the mill were very frequently attacked by pneumonia, especially before certain precautions were taken to prevent the excessive development of dust.

The Thomas phosphate slag contains, in addition to 24% iron protoxid and small quantities of oxid of iron, about 8% of free unslaked lime, 40% of phosphate of lime, and 30% of silicate of lime; but, according to his belief, neither the chemical action of the quicklime nor that of the phosphate of lime had anything to do with the production of the disease. Nor does he pretend to say whether any injurious influence is to be ascribed to the silicate of lime which is very readily decomposed, even with carbonic acid, and when thus broken up forms a gelatinous mass similar to lime which, mixed with mucus, might easily exclude the air from porous bodies like the alveoli of the lungs. He is rather inclined to seek the deleterious properties of the pulverized slag in its morphology, in "the tendency of the smallest dust particles to adhere mechanically [to the tissues. —Ed.] and to set up a constant scratching and irritation."

Enderlen has made experimental investigations of the effects of Thomas phosphate meal upon the lungs, in connection with an examination of the lungs of two men who died of pneumonia caused by the inhalation of Thomas slag-dust. In the first of these cases he found in the cover-glass preparations, taken from the right pneumonically affected lung, lanceolate cocci which were provided with capsules. Mice died when inoculated with the pulmonary fluid; numerous lanceolate cocci with capsules were found in the blood, in the liver and spleen. When treated with iodid of potash, they were not decolorized. Therefore they were Fraenkel-Weichselbaum cocci. The sections showed the typical picture of croupous pneumonia; numerous pneumococci were present in the alveoli, which were densely filled with fibrin. In cover-glass preparations of the second case, in which also the right lung was affected, bacilli were found in addition to pneumococci. In the sections again pneumococci could be demonstrated by Weigert's fibrin method.

His investigations of the consequences of experimental inhalations of Thomas slag-dust showed that it causes an irritation of and injury to the pulmonary tissue consisting of an interstitial inflammation which temporarily abolishes the function of several alveolar groups. He assumes that the loss of function may become permanent in a long duration of the experiment by the shrinking of the tissue which forms in the interstices. His failure to produce the disease that he was trying to produce,—namely, croupous pneumonia,—even when the inhalation was followed by an injection of pneumonia-juice into the rabbit's trachea, may be attributable to the fact that pneumococcus infection in animals is followed by septicemia and not by a local reaction. In the case of man, however, he assumes that the dust of Thomas phosphate slag acts both mechanically, by reason of the sharp, angular constituents, and chemically, by reason of the caustic (unslaked) lime, and the injury to the pulmonary tissue thus produced provides a favorable soil for the multiplication of the pneumococci.

Loeb, too, had the opportunity of examining a considerable number of rabbits which were for a longer or shorter time exposed to an atmosphere of Thomas phosphate meal-dust. He came to the conclusion that the pneumonia produced in these animals was not originated by bacteria, but by the partly chemical, partly mechanical, influence of the phosphate meal. Within the affected parts multi-nucleated cells in large quantities were present in the pulmonary parenchyma as well as between the alveoli. They were of an irregular form and contained dust or iron particles. Their nuclei were ranged against the walls and were well stained with basic stains, contrary to the central part of the cell, which preserved a yellow color. At the edges colloid masses projected here and there in the form of vesicles. The whole condition showed some resemblance to the changes in genuine desquamative pneumonia as reported by Buhl.

In view of the importance for the pathogenesis of dust inhalations of the changes produced, I have also made experiments with Thomas phosphate meal. Dissolved in water, it was injected into the trachea of rabbits with a Pravaz syringe. I shall report the results later on at the proper place, and shall now discuss a very important point in pneumonokoniosis—namely:

THE ENTRANCE OF DUST INTO THE PULMONARY TISSUE.

To-day we may consider the fact to be finally proved that inorganic dust particles suspended in the air may penetrate into the pulmonary tissue and remain there. It will not be the question of organic dust particles or such as consist of dead organic substances, the inhalation of which belongs mostly to the sphere of the already described aspiration pneumonia. There cannot be any more doubt that the quantity of the dust particles inhaled must be proportionate to the quantity of dust in the air; hence the kind of dust which is present in the lungs depends upon the individual's occupation and the formation of dust connected with the same. It remains now to discuss and to prove the manner in which the dust enters the lungs and deposits itself there, and whether and how it penetrates from there into the pulmonary tissue.

Villaret is the only one who upholds the theory that the coal-dust existing in the lungs is not inhaled, but is brought into the lungs and the bronchial glands from the digestive tract with the circulating blood of the portal vein. The experiments referring to this cannot very well be regarded as convincing. None of the later investigators agreed with him. They all agree that the dust is inhaled through the larger bronchi, finding its way thence into the lungs. Traube is of the opinion that the particles, driven in by the inspiratory air-current, reach the alveoli with a certain velocity and perforate the opposite cell-walls. Kuborn simply says that the coal particles perforate the alveoli of the lungs, penetrate into the interstitial tissue, and from there are admitted into the lymph-vessels and deposited in the lymph-glands. Lewin merely mentions that the coal particles succeed in getting into the lungs mechanically.

In regard to the motive force that enables the particles of coal-dust to penetrate the pulmonary tissue, Rindfleisch believes, in view of the improbability, not to say impossibility, of the particles of coal detaching themselves again from the alveolar wall, that they are carried off by the extravascular nutritive juices (the ultimate lymph-stream) and eventually enter the lymphatic stream. In their passage through the lymphatics they occasionally meet cellular elements which possess the property of permanently fixing small, solid bodies in their protoplasm. These elements include both the spindle-shaped connective-tissue cells, and the wandering, ameboid cells which are also present in the pulmonary tissue, and carry the black pigment that they have absorbed with them wherever they go. The remainder of the pigment, that which has not been absorbed by cells in its passage through the lymphatics, is carried to the root of the lung and enters the mediastinal lymph-glands.

Direct injury of the tissue by the entering dust particle is out of the question, as Hirt correctly argues from the fact that not only sharp, angular bodies, like particles of charcoal, for instance, but also spherical, perfectly harmless particles of oxid of iron, for instance, are found in the alveoli. The present state of our knowledge, he says, compels us to adopt the explanation that the tissue-elements are merely forced apart by the entrance of the dust particles, without being injured, and at once unite again and close the momentary opening after the foreign body has passed through—a mechanism to which the term "penetration" has been applied by Robin.

Knauff believes that the passage of particles of coal-dust from the surface of the alveoli into the parenchyma necessitates the assumption of a free communication, which, however, cannot be demonstrated objectively. This path, whatever it may be, is followed not only by the free particles of coal-dust, but also by the epithelial cells that carry carbonaceous pigment and enter the deeper layers of the tissue. These cells enter the lymphatic system, and eventually reach the lymph-glands by way of the lymphatic trunks. Some of them, however, escape from the lymph-stream and enter some cleft in the tissues outside the lumen of the lymphatic trunk, where they stick fast.

According to Buhl, the deposition of various kinds of dust—such as iron, coal, silicum, and lime—not only in the pulmonary, but also in the laryngeal and bronchial tissue, where it is also found at times, is most readily explained, especially in the case of the pulmonary tissue, on the assumption that the alveolar epithelium represents a layer of lymphatic endothelium lining the inner surface of the alveolar wall. If the alveoli are regarded as lymph-spaces, it is possible to suppose that aspirated dust particles are carried along by the tissue-juices, or the movable cells contained in it, and deposited by the way or at some remote point.

Stavjansky injected an emulsion containing cinnabar, ultramarine, indigo, and coal into the lungs through a tracheal wound. The pulmonary alveoli contained a moderately large number of cellular elements, the majority of which resembled white blood-corpuscles, and were filled with cinnabar granules; but the alveolar epithelia also contained cinnabar. That these cells were white blood-corpuscles he concludes from the fact that in synchronously injecting cinnabar into the blood through the jugular vein, and indigo into the lungs through the trachea, he found cells in the alveoli which contained cinnabar and indigo at the same time.

Von Ins is very positive in maintaining that all the cinnabar particles which are in the alveoli are absorbed by white blood-corpuscles and carried into the pulmonary tissue from the alveoli, passing through the projecting edges which separate the entrance of the different alveoli into the infundibula, exactly at the junctions of these edges. Most likely there are open passages which lead from the alveoli into the stroma.

Ruppert deduces from his experiments the diametrically opposite conclusion that the dust in the alveoli is in part taken up by epithelial cells, and in part enters the tissue in the free state, just as it was inhaled—*i. e.*, without being contained in cells. Transportation by ameboid cells, if it occurs at all, must be very limited. The presence of cells containing dust in the tissue of the alveolar septa is simply an artefact due to incorrect methods of investigation.

Schottelius, again, assumes a participation of the alveolar epithelium, and of the white blood-corpuscles which had entered the alveoli, in the absorption of the dust, and believes that the latter are responsible for carrying the dust into the tissue.

Arnold, after numerous experiments and observations, also believes that the inhaled dust gets into the pulmonary tissue either as such,—*i. e.*, without being bound to cells,—or is absorbed by lymphoid, or even by epithelial cells and transferred into the stroma. While but few dust particles are found in the alveolar wall, they are more plentiful in the inter- and peri-infundibular connective tissue, and most abundant in the peribronchial and perivascular connective tissue. But the deposition of dust does not always occur in the direction of the tissue-juice and lymph-current. If one considers that most of the dust generally lies within the alveoli, from where it can be transferred only to the lymphatic vessels through the system of lymph clefts, the presumption seems to be most correct that the dust, which accumulates in the neighborhood of the lymphatic vessels, or at least most of it, has been brought there by the lymph clefts and not by the lymphatic vessels.

It is very remarkable that, in all experiments attempting to explain the introduction of dust into the lungs chiefly or exclusively by the importation from the alveoli, with or without intervention of the cells, one fact has been almost entirely overlooked: the size of the dust-corpuscles which are found in the pulmonary tissue. Only Merkel takes this fact into consideration, and concludes from it that the entrance of dust particles cannot be effected exclusively by cells; for one finds splinters principally of charcoal, so large as to preclude a transportation through cells, at places of the pulmonary tissue which correspond neither to obliterated alveoli nor to obliterated bronchioles.

I am in a position to confirm this observation, and to add that dust particles are occasionally found within the pulmonary tissue which even exceed the alveoli in size, and cannot therefore have entered the pulmonary tissue by way of the alveoli, but must have penetrated the wall of a bronchus. How the passage through the bronchial wall is effected is still a mystery, and this is probably the reason why the fact itself has never received the attention it deserves. The explanations offered by Traube and Lewin cannot be accepted as final.

Another fact of essential importance for explaining the processes in dust inhalation, which has not received the attention it deserves is the occurrence of pigment—*i. e.*, of dust—in the costal pleura. Virchow was the first to demonstrate the presence of pigment at this place, and used it in support of his earlier opinion that black pigment in the organism originates from hemoglobin. He says: "The character of this pigment has given rise to a great deal of argument, which would not have been necessary if it had only been ascertained that the black pigment is found between the elastic fibers in the parenchyma proper of the lungs and even outside of the pulmonary lobules in the connective tissue that forms the septa: in fact, that it exists not only in the lungs, but sometimes in the costal pleura as well."

Furthermore, it was demonstrated by experiments that corpuscular elements entering from the outside may penetrate into the costal pleura through the respiratory tract. Fleiner reports an anthracosis which was produced experimentally by Arnold, and in which the pseudomembranes between the very anthracotic parietal and visceral layers of the pleura contained large accumulations of coal, such as to leave no doubt that the coal was transferred from the pulmonary to the costal pleura by the agency of the pseudomembrane. His own experiments further proved the fact that in a few minutes' time corpuscular elements are taken up from the pleural cavity and forwarded in the lymph-passages of the pleura, or deposited in the lymphatic apparatus and regional lymphatic glands. But the pseudomembranes with their numerous newly formed lymph-passages are not the only avenues for the migration of inhaled dust from the pulmonary alveoli into the pleura. Fleiner assumed that the pleural and subpleural lymph nodules described by Arnold secrete the dust in the pleura. In the autopsy of elderly persons it can be demonstrated that coal particles are carried from the surface of the lungs to the costal pleura solely by bands of pseudomembranous connective tissue.

Grawitz has made some valuable investigations in connection with the mechanism of dust-inhalation. Selecting a large number of young individuals, who had not been particularly exposed to a dust-laden atmosphere during life, he made microscopic examinations of the costal pleura and found in it three kinds of pigment which proved to be (1) particles of coal-dust, (2) iron-dust, and (3) amorphous derivatives of hemoglobin. In the hope of learning how the particles of dust had reached the costal pleura, he insufflated charcoal-dust, iron-dust, and cinnabar into the lungs of rabbits through a tracheal wound, and found scattered particles of dust in the costal pleura as early as twenty-four hours after the insufflation. To decide the question whether these particles reached the pleura from the hilum of the lung, he produced a pneumothorax on the animals, and now blew the same kinds of dust into the trachea. On the side of the pneumothorax the parietal layer of the pleura did not show a trace of pigment deposit. He therefore concludes, the same as Fleiner, that an actual migration of dust particles takes place from the pulmonary to the costal pleura. I repeated these experiments, injecting with the Pravaz syringe cinnabar solutions—which I first filtered through linen to detain larger particles—into the unopened trachea. I found in a rabbit which was killed two days after the injection, besides small areas of cinnabar in the lower lobe, two accumulations of cinnabar below—*i. e.*, outside of the costal pleura. One was as large as a pinhead; the other, close by, had the form of a line about 3 mm. long. But there were no adhesions between the costal and the pulmonary pleura.

Supported by the material on hand, I shall try to give an explanation of the processes in dust inhalations which shall do better justice to the facts than has heretofore been done.

An adequate explanation is still lacking for the well-known fact that comparatively few particles of dust are found in the alveoli of cadavers, either in human subjects who have been exposed to an atmosphere charged with the corresponding variety of dust, and have therefore inhaled it, or in animals in which an artificial pneumonokoniosis has been induced.

Another circumstance that has never been explained is that, although most authorities assume a direct transportation of the dust particles from the alveoli into the pulmonary tissue, the least dust is found in the interstices between the alveoli,—according to some authors, they are entirely free from dust,—while it is found in abundance in the interlobular, and even more plentifully in the peribronchial and in the perivascular tissue, and always outside of the cells.

Finally, how can it be explained that inhaled dust particles penetrate through the pulmonary pleura into, and even below, the costal pleura?

Regarding the first point, we may start with the fact that dust-molecules are absorbed by the protoplasm of cells which are present in the alveoli. Probably this is done with all particles that get into the alveoli and are not larger than the cells themselves. In human lungs, at least, I have always found in the, I may say, very numerous instances of anthracosis under the most varying pathologic conditions, that coal-corpuscles within the alveoli were always inclosed in cells. Only in rabbits, into the mouth of which coal mixed with glycerin was put after bilateral vagotomy, I found coal particles which were larger than the cells themselves outside of the cells in the alveoli.

Coal particles in human alveoli are almost always of a uniformly small size. This is very easily explained by the fact that only the smallest and lightest particles can be drawn into the alveoli by the air-current, while the larger and heavier ones must remain on the mucous membrane of the larger and smaller bronchi.

The cells in the alveoli which are more or less filled with dust, the "dust-cells," are of considerable size, some with, others without, nucleus, and correspond in every respect to alveolar epithelium. Their size alone speaks for the correctness of this contention. Another proof is the finding of such dust-cells in alveoli which are more or less filled with white blood-corpuscles by pathologic processes. I have seen cases of chronic as well as of desquamative pneumonia in which, within the alveoli and among the very numerous white blood-corpuscles, coal-containing cells were present which by their size differed from leucocytes. In none of these cases was a single coal particle found in a white blood-corpuscle.

I once succeeded in obtaining a very instructive illustration of the part played by the alveolar epithelium in the deposition of dust particles by injecting a mixture of Thomas phosphate meal and water into the trachea of a rabbit according to the method described on page 666, and killing the animal four days after the injection. The entire alveolus, as shown in figure 10 on Plate 7, is filled with small round cells, while only large cells, the identity of which with alveolar epithelium is unmistakable, contained dust particles.

I therefore feel compelled to agree with those authors who assert that the white blood-corpuscles or ameboid cells have little or nothing to do with the deposition of dust particles. The theory that the dust particle is carried by a migratory cell from the alveolus into the pulmonary tissue thus loses an important prop.

In view of the following facts, namely, that the alveolar septa by common consent contain only insignificant masses of dust, or none at all, according to some authorities; that, according to Ruppert's experiments, the occurrence of dust within the alveolar septa in experimental pneumonokoniosis is an artefact; and that, according to my own observation, in the rare but undoubted cases in which particles of coal are found in the alveolar septa in man the pigment is invariably free and is never found within the cells, the assertion that the presence of dust in the pulmonary tissue is the result of transportation by means of cellular structures from the alveoli must be declared unproved.

As long as the corresponding bronchi are intact, the dust which is inhaled into the alveoli and absorbed by the alveolar epithelium is mostly expectorated with these cells. Whoever has an opportunity of making clinical observations will confirm the fact that cells filled with coal-dust are very often found in the sputum, even in cases in which no material accumulation of coal is found in the lungs post-mortem.

These considerations, which speak against the transportation of dust-particles from the pulmonary alveoli into the pulmonary tissue, are made weightier by another fact—namely, the finding in the peribronchial and perivascular tissue of larger particles of coal, iron, and phosphate of lime which is contained in Thomas phosphate meal. Such large particles of dust as are here present do not occur in the alveoli at all, so that it will scarcely be necessary to remark that they are much larger than the cells of alveolar epithelium, and cannot therefore be absorbed by them and carried into the pulmonary tissue.

But I am able to add to these negative proofs another, to my opinion, indisputable one, which compels us to admit the fact that large dust particles enter the pulmonary tissue through the bronchial wall. Any speculations in regard to the motive power must be based on this fact.

Thomas phosphate meal dissolved in water was injected into the trachea of a rabbit and the animal killed after four days. The microscopic examination showed, besides other changes which will be referred to later on, the presence of large particles of phosphate of lime in the bronchial wall and outside of it in the peribronchial tissue. This fact is plainly shown in figure 9 on Plate 6. While the finding in the peribronchial tissue of other kinds of dust, principally coal-dust, is not accompanied by any changes in the tissue which could be utilized to decide the question whether the particles were brought from other parts of the pulmonary tissue or whether they penetrated at the same place, the presence of phosphate of lime is followed by inflammatory changes which prove conclusively that these particles cannot have taken any other way but that through the bronchial wall. In figure 9 the foreign bodies are surrounded by granulation cells which form a small peribronchial nodule. The inflammatory properties of the foreign body prove, therefore, that it must originally have entered at the place where it was found, and could not possibly have gotten there from any other place in the pulmonary tissue. A small number of granulation cells visible on the mucous membrane of the bronchus, and a defect in the layer of smooth muscle membrane in the bronchial wall, clearly indicate the place of entrance.

The mechanism of the passages of foreign bodies through the bronchial wall is also readily explained by a reference to the figure referred to. The smooth musculature of the bronchial wall in the rabbit, as the figure shows, consists of a circular and of a longitudinal layer of smooth muscle-fibers. Instead of being superimposed one upon the other, these layers interlace like the web and woof in a piece of cloth. The circular layer is more robust than the longitudinal.* Wherever the large granules of calcium phosphate in the peribronchial tissue are surrounded by nodules of granulations, a separation of the circular muscle-fibers is observed, while the longitudinal fibers disappear and are replaced by processes emanating from the granulation nodules, which also consist of granulation cells and extend to the epithelium on the inner surface of the bronchus. The conclusion is obvious that the foreign body in its passage through the bronchial wall has divided the longitudinal muscle-fibers.†

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*If it were to be shown that the same conditions existed in the bronchial musculature in man,—*i. e.*, that there was a layer of longitudinal, and a more robust layer of circular smooth muscle-fibers, interwoven one with the other, a very plausible explanation of the origin of asthma in man would be possible. The preponderance of the circular muscle-fibers must, in all cases in which the function of the involuntary muscles is disturbed, lead to a contraction of the smallest bronchioles, which under normal conditions is prevented by the action of the longitudinal fibers.

† It must be specially mentioned that all these changes have been demonstrated according to the method of preparation mentioned on page 396, principally by means of the Biondi-Heidenhain solution.

EXPLANATION OF PLATES 6 AND 7.

FIG. 8.—Cross-section of an artery in syphilitic pneumonia of the new-born. Marked increase in the cellular elements of the adventitia, the thickness of which is equal to the width of the lumen. A smaller vessel, seen in longitudinal section, shows the same change. The whole is contained in a large, empty lymph-space. × 390 (Leitz objective 6, ocular 3).

FIG. 9.—Longitudinal section of a bronchiole from a rabbit killed four days after an injection of Thomas's phosphate meal into the trachea. At the upper and lower ends of the bronchiole the layer of smooth muscle is visible, the more robust transverse fibers interlacing with the more slender longitudinal fibers. At the center of the bronchiole, which is copied exactly from nature, the underlying alveoli are seen. On the left, the bundles of muscle-fibers, which are arranged concentrically and appear in transverse section, are intact; on the right they are interrupted, and surrounded by a large focus of granular material containing several particles of Thomas's phosphate meal. × 85 (Leitz objective 3, ocular 3).

FIG. 10.—Alveolus from the lung of the same rabbit that furnished the specimen for Fig. 9. The alveolar epithelium is filled with fine granules of Thomas's phosphate meal of uniform size. × 800 (Leitz oil-immersion $\frac{1}{12}$, ocular 3).

FIG. 11.—Two pulmonary alveoli from a case of chronic pneumonia. The masses of newly formed connective tissue are well supplied with blood-vessels, and communicate with one another as well as with those belonging to adjacent alveoli; the same conditions are seen in the projecting portion on the right-hand side in the figure. × 275 (Leitz objective 6, ocular 1).

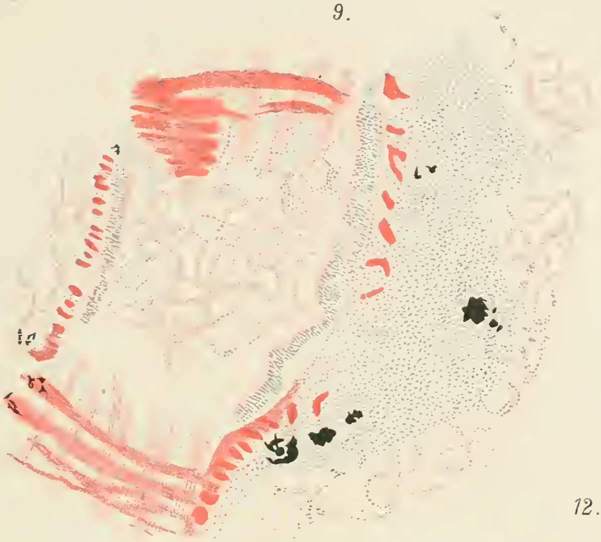
FIG. 12.—Parasitic (?) structures from a diffuse lobar pulmonary sarcoma. × 800 (Leitz oil-immersion $\frac{1}{12}$, ocular 3).

FIG. 13.—Fibrinoid degeneration of the connective tissue in the adventitia of a pulmonary artery, after thrombosis of the vessel following congestion from cardiac insufficiency. The swollen fibers of connective tissue are seen partly in longitudinal and partly in transverse section. × 390 (Leitz objective 6, ocular 3). The drawing includes two microscopic fields.

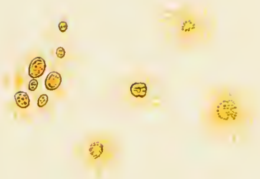
The specimens are all stained with Biondi-Heidenhain's three-color mixture.

PLATE 6.

9.



12.



11.

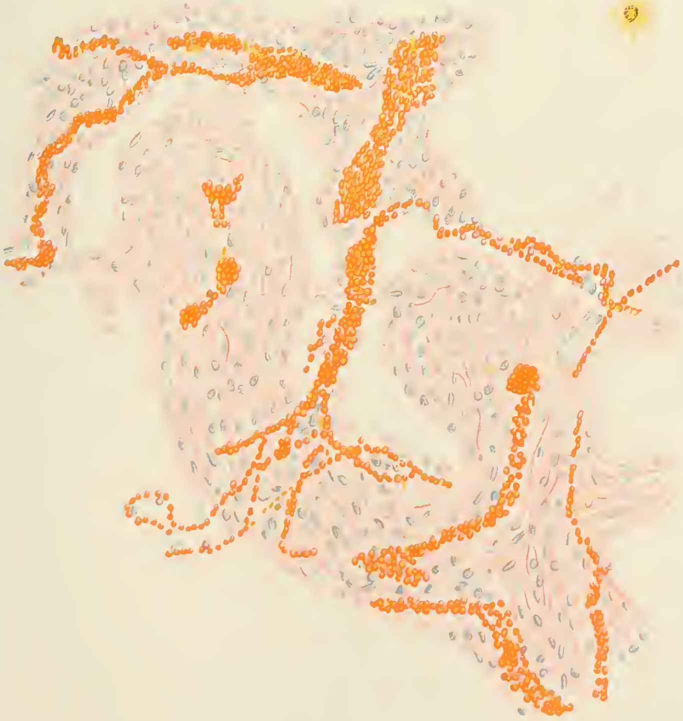
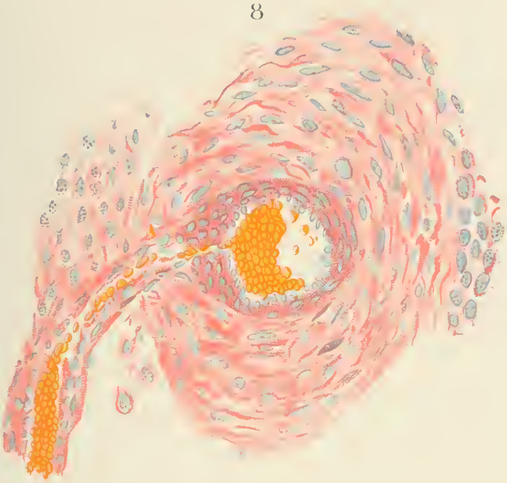


PLATE 7.

8



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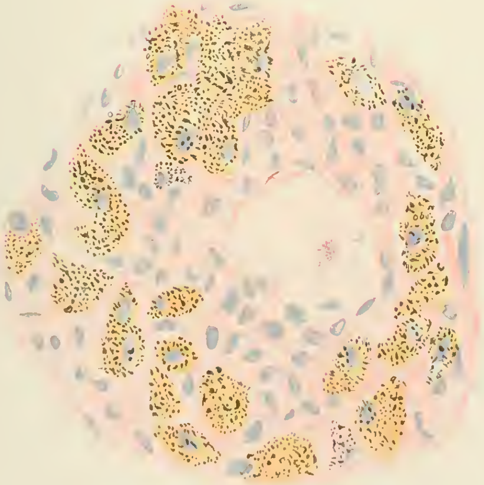

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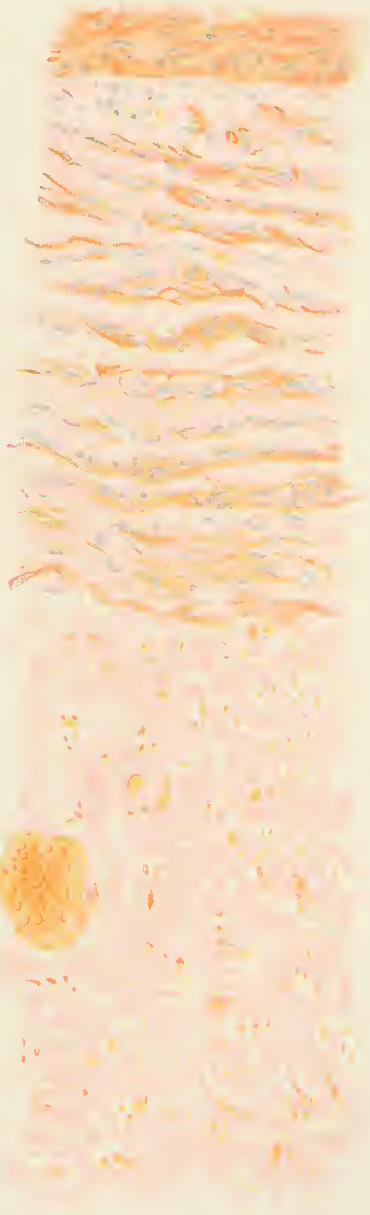

I must leave it to future investigators to decide whether the lymphatic nodules which were found by Knauff, von Ins, Arnold, and Heller, when investigating the changes caused by the inhalation of dust, may not be in part regarded as newly formed areas of granulation cells in the neighborhood of or surrounding foreign bodies.

After this explanation there can be no more doubt that the principal share of introducing dust into the pulmonary tissue is done by the finer bronchi. There remains only to explain the mechanical process which enables the dust to penetrate the bronchial wall. That any active share of the bronchial mucous membrane or its elements is out of the question is proved by the fact that there are no dust particles to be seen within the cells of the mucous membrane, and that the particles are mostly larger than the cellular constituents of the finer bronchi.

Since the air-passages remain absolutely passive, it was supposed that the particles, driven by the air-current, penetrate the tissue (Traube), or, more plainly, simply mechanically pierce the pulmonary tissue (Kuborn, Lewin); which tells us nothing. If it be even admitted that some kinds of dust particles are sharp and therefore adhere more readily to the bronchial wall, this would only render it more difficult to expectorate such corpuscular elements. For part of the dust is certainly expelled, as can best be proved experimentally. If cinnabar is injected through the trachea into the lungs of a healthy rabbit, later on such a small part of the cinnabar is found in the lungs that a considerable amount must have been expectorated.

Just as little will it be correct to consider the presence of a bronchial catarrh as a factor in the entrance of coal-dust into the tissue, in spite of favorable conditions caused thereby for the depositing of dust-corpuscles.

The physical and chemical properties of the substances inhaled at most act as predisposing by injuring the mucous membrane and thus enabling the foreign body to cling more tenaciously.

The penetration of corpuscular elements into the pulmonary tissue is caused solely by the negative atmospheric pressure which exists in the thoracic cavity. We know that the thoracic wall forms with the diaphragm an air-tight space which is adjacent to the pulmonary tissue, and that the latter is dilated by the expansion of the thoracic cavity. In every breathing pause the muscles of the thorax and the diaphragm are opposed by the centripetal pull of the pulmonary tissue. Therefore in the resting thoracic cavity there exists a negative pressure, caused by the elastic tissue of the lungs. Donders demonstrated this pressure to be 6 mm. of mercury, by hermetically closing the trachea by a manometer when experimenting on a cadaver, and then opening the thoracic cavity by puncture. During inspiration a heavier strain is necessarily thrown on the elasticity of the pulmonary tissue, so that the negative pressure in the thoracic cavity is increased. Therefore the pulmonary parenchyma will be in the position of an elastic membrane which is drawn over the opening in the glass cylinder in an air-pump, while the pressure of the air in the glass cylinder is reduced to 6 mm. of mercury. With every inspiration the lung experiences a stronger strain, exactly like the elastic membrane over the opening of the cylinder of an air-pump if the rarefaction of the air is increased. Now, if we transfer this process to the mucous membrane of the lungs covered with dust particles, it is evident that during inspiration the mucous membrane must first of all become distended, consequently its elements must be further apart than during expiration; and, secondly, against the increased negative pressure in the thoracic cavity a proportionately increased positive pressure in the bronchi must exist, which, according to the laws of physics, must influence the small dust particles and force them during inspiration into the distended, slightly yielding tissue.

This physical process offers the only plausible explanation for the appearance of pigments beneath the costal pleura when no pleural adhesions are present. If the pigments are inhaled into peripheral bronchi,—*i. e.*, bronchi situated near the surface of the lung,—they follow the inspiratory air-current to the point where the negative pressure originates, as in an air-pump; that is, they enter the intercostal musculature.

It follows from what has been said that two processes are exclusively, or at least chiefly, concerned in the deposition of dust particles in the pulmonary tissue. In one of these processes the smallest particles of dust are carried by the air-current into the alveoli, where they are taken up by alveolar cells and later, for the most part, expectorated along with the cells. In pathologic conditions, especially when an exudation of white blood-cells into the alveoli takes place, the dust-cells remain among the cells of the exudate. In the second process larger particles of dust escape from the inspiratory air-current and deposit themselves on the mucous membrane of a smaller bronchus, from which they are not expelled by the ciliated epithelium if the physical or chemical properties of the foreign body are such as to favor its clinging to the mucous membrane, or the functional activity of the ciliated epithelium has been impaired by pathologic conditions, particularly catarrh. In the latter event the dust particles are drawn into the pulmonary tissue during the increase of the negative pressure within the thoracic cavity which takes place during inspiration, the entrance of the particles being facilitated by the inspiratory distention of the tissues.

PATHOLOGIC ANATOMY.

It is impracticable to discuss the pathologic alterations produced by the inhalation of dust in such a way as to include all the varieties of the noxious material; although the present discussion, in one respect at least, is based on a common characteristic of all the various kinds of dust, since we are dealing exclusively with inorganic substances or the ash of organic substances. In the case of organic substances the pivotal point of the discussion, the mode of entrance into the pulmonary parenchyma, is of no importance.

Schottelius' assertion that inorganic dusts, which exert a purely mechanical influence, even when introduced in large quantities, are incapable of producing profound destruction of the pulmonary tissue, appears to me too sweeping; I am rather inclined to agree with Lancereaux, who says: "Not all kinds of dust exert an equally deleterious influence on the pulmonary tissue."

The inspiration of coal-dust is the least injurious. Traube, on the strength of his above-related observation, and Maurice deny any direct injurious effect. Coal-miners may enjoy the best of health, even after twenty years' occupation in the mines, although their lungs are pigmented black, as is proved by autopsy and by the color of the sputum. On the other hand, pathologic processes in the lungs favor the admission of coal, as is shown by the reported case of miliary tuberculosis. The coal-dust was most plentiful around the nodules. The latter diminished the elasticity of the pulmonary vesicles in their immediate neighborhood, and so favored the deposition of the inhaled coal particles, on one hand, and, on the other, prevented their expulsion.

While Greenhow reports only a single case of anthracosis, Seltmann explicitly discusses its consequences for the pulmonary tissue, and comes to the conclusion that anthracosis of lesser degree may keep up and promote certain diseases of the respiratory organs which had developed before its appearance, but it does not produce any specific pathologic conditions, certainly no inflammatory processes of an acute or chronic kind. The pulmonary substance never shows any consolidation of the interstitial tissue, even when coal-dust is deposited in large quantities. According to his opinion, coal-dust has a favorable influence in tuberculosis. The scarcity of this affection among the youngest workmen, and more so its extreme scarcity and conspicuously slow course among miners who are twenty-five or thirty years of age, will not allow of any doubt. Some authors are so convinced of the correctness of this view that they recommend the inhalation of coal-dust, and have actually had it inhaled. Nevertheless, Seltmann does not deny the possibility that the pressure exerted by large accumulations of coal-dust is apt to cause independently a very limited inflammatory process, which later on, in consequence of the general poor nutrition and the special disturbances of nutrition of the lungs, may lead to the formation of abscesses and cavities.

Merkel also says that substantive affections of the pulmonary tissue are relatively rare as a consequence of the inhalation of coal-dust.

Crocq even opines that coal is not only relatively not injurious to the lungs, but effects an antagonism against tuberculosis. This is made apparent by the rare occurrence of the latter in coal-miners and the appearance of coal in the neighborhood of obsolete or healed tubercles.

According to Fripier's report, which is based on pathologic observations and experimental investigations, phthisis of miners is only a phthisis with predominance of the fibrous form in which the infiltration of the pulmonary tissue with coal, especially in the diseased areas, may be so plentiful that the organ in consequence has a peculiar appearance which sometimes renders difficult the demonstration of tuberculous alterations.

In view of my own anatomic investigations I may absolutely concur in the opinion of the relative innocuousness of inhaled coal-dust particles. The microscopic examination of lungs of elderly people who had been employed in an atmosphere of coal-dust,—for instance, coal-carriers, firemen,—sometimes reveals along the bronchi and blood-vessels such dense deposits of coal that the peribronchial, and even the perivascular, tissue may be entirely covered, but no inflammatory changes exist. The interstitial tissue is not widened, the neighboring alveoli are intact.

This statement appears to conflict with the well-known fact that extensive areas of black induration are particularly apt to be encountered in the upper portions of the lungs. Friedreich has emphasized this fact, chiefly, it is true, in connection with the question of the origin of the pigment. Since large foreign bodies, when inhaled, preferably lodge in the lower lobes of the lungs, especially on the right side, he says it is fair to assume a similar tendency on the part of small particles of dust that have been drawn into the lungs. Why is it, Friedreich asks, that the parenchymatous change known as black induration does not predominate in the lower lobes in the case of these workmen? Why, on the contrary, are the upper lobes, and particularly the apices, the seat of black induration? This is the more remarkable when it is remembered that the diaphragmatic and intercostal type of breathing predominates in men. He explains this phenomenon by assuming a special irritability and local predisposition to certain diseases on the part of the pulmonary apices.

Hanau also explains this process by a predisposition of the pulmonary apices, referring to this report of Friedreich, as well as to Kussmaul's case of chalicosis and Zenker's cases of siderosis. The apices, according to his opinion, inspire very well and are therefore well able to inhale dust and micro-organisms, but they do not expire well, and therefore the inhaled particles have an opportunity of remaining in them; in fact, they may be driven further into the tissue by an inverse air-current and attach themselves to the inner surface of the alveoli.

I myself, when discussing the conditions which predispose to phthisis of the lungs, attributed the peculiar predisposition of the pulmonary apices to the construction of the thorax which does not permit as complete inspiratory distention in the apices as in the other pulmonary sections, so that the inspiratory and expiratory current is not able to overcome any obstacle in the finer bronchi. Such an obstacle may be formed by secreted mucus, cast-off epithelium, exudative cells in croupous pneumonia, and thereby the material may be given for the colonization of tubercle bacilli and for the instigation of further inflammatory processes, resulting in tuberculosis.

But that the pulmonary apices should inspire well but expire badly, as Hanau presumes, is not quite in keeping with our physical views of the efficiency of the elastic pulmonary tissue.

This is also contradicted by the fact that in all cases of coal deposits in otherwise healthy lungs the most plentiful peribronchial infiltrations of coal are not by any means found in the apices.

If we add that, according to the opinion of by far the greatest number of authors, inhaled coal-dust is not the direct cause of pulmonary diseases; and that, according to my above explanation, catarrhal conditions of the bronchial mucous membrane especially favor the deposition and subsequent penetration of dust particles,—the predominant frequency of black induration in the upper lobe may be explained by the existence of a focal disease which preceded the induration or set in independently of it, and in the neighborhood of which catarrhal and further reaching inflammatory processes occur. These latter, on one hand, favor the remaining of the inhaled coal particles, and on the other, in consequence of a possible irritating effect of the coal on the pathologically changed sections of the pulmonary tissue, lead to a fibrous consolidation which otherwise would not have taken place.

Therefore the answer to the above questions of Friedreich is that black induration occurs most frequently in the pulmonary apices, because these are most frequently the seat of pathologic processes.

This explains the possibility of firm encapsulation of tuberculous foci in the pulmonary apices, and thereby the prevention of an advancing tuberculous disintegration of the pulmonary tissue. Probably the above-mentioned rarer occurrence of tuberculosis is to be ascribed to this fact.

Consequently the assumption that anthracosis in itself may lead to the formation of cavities may be rejected. This supposition is, in the first place, contradicted by the fact that healthy pulmonary tissue remains intact in spite of abundant inhalation of coal-dust, and, secondly, by the just demonstrated possibility of a limitation of tuberculous foci and the prevention of advancing disintegration of the pulmonary tissue by fibrous consolidation of the same. The cavities unquestionably originate in a disintegration of tissue which precedes the black induration. But, at the same time, the occurrence of acute tuberculous processes with already existing anthracosis is not impossible. Maurice observed such a case in a laborer, forty years of age, which terminated fatally within four weeks with rapid formation of cavities and black sputum.

However, it is to be mentioned that the same author ascribes the rarity of chronic phthisis in coal-workers not to the effect of coal-dust, but to the fact that only few predisposed individuals would choose this laborious occupation. I should like to add: Is it not likely that a considerable number of men who, during their employment as coal-workers, develop chronic tuberculosis give up this trying occupation and are lost sight of for purposes of statistics?

Of greater importance for the origin of morbid changes in the pulmonary tissue are the various kinds of stone-dust and steel-dust. Crocq says: Coal-dust is almost harmless in comparison with stone-dust in stone-cutters, and metal-dust in the manufacture of needles, in steel-filers, in metal-grinders, etc., who rarely grow older than forty years. In his experiments he introduced oxid or bichromate of lead with a solution of starch into the trachea, and in a short while produced lobular pneumonia, and even tubercles, while animal- or wood-charcoal or soot were deposited in the lungs and in the bronchial glands without causing any further organic changes of the pulmonary tissue.

Arnold proved, by his experiments, that inhalations of emory and sandstone, which were followed much more rarely and after a longer interval by the filling of the alveoli with dust, caused more extensive inflammatory infiltrations than the inhalation of soot and caused them earlier. After soot-inhalation the foci are fewer, and only exceptionally are produced early.

Merkel, referring to 16 observations, exhaustively reported the pathologic changes in human beings that are caused by the inhalation of iron-dust. According to the kind of dust inhaled the lungs are either black (magnetic oxid of iron and phosphate of iron) or red (oxid of iron). That portion of the parenchyma which still contains air is traversed by hard, cicatricial bands which constrict individual portions of the tissue and cause them to project in the form of nodules both on the cut section and on the superficies of the lung. A characteristic feature is the presence throughout the tissue of numerous extremely hard nodules, varying in size from that of a hemp-seed to that of a pea and over, which grate under the knife and on section appear grayish-yellow with punctiform and linear markings. Many of these nodules contain a distinct lumen as large as a pin-point. Sometimes the nodules coalesce. Cavities in the apices and caseous foci also occur.

After giving this description Merkel remarks: "While processes of this kind are rare in the lungs of coal-workers, they appear to be constant in the lungs of iron-workers attacked by pneumonokoniosis." Whether the difference is due to the chemical inertia of coal as compared with metallic dust, or to the size and shape of the dust-molecule,—the molecules of charcoal are usually larger than those of metallic dust,—he does not pretend to decide.

According to Merkel, the pathology of chalicosis agrees with that of siderosis. There are black, centrally whitish nodules, as large as a flax-seed to a pea, which project above the level of the cut surface, are more coarse, and grate more under the knife than those in siderotic lungs. It is only remarkable that these nodules never attain the same size as the nodules in siderotic lungs, nor coalesce to any extent. Chronically indurated parts in stone-cutters' lungs are mostly of uniform structure, and never show any evidence of being formed by the coalescence of single nodes.

While all and every anatomic change taking place in consequence of inhalation of dust of coal, iron, and sand, or of a mixture of these kinds, proves to be a process of a chronic character, one kind of dust, the production of which for agricultural purposes has grown to be of great industrial importance, enjoys the sinister privilege of being able to cause acute inflammations of the lungs—namely, the Thomas phosphate meal.

Of 20 patients of Ehrhardt who were affected by the inhalation of Thomas phosphate meal, 6 died, and in 4 cases he could hold an autopsy. The disease principally attacked the lower lobes. The latter appeared very congested and showed a uniform smooth consolidation in which only a few smaller foci had a granular cut surface. Only in one instance the lower lobe was uniformly gray, infiltrated, and slightly granulated. The pleural covering of the affected parts showed fibrin layers and sometimes hemorrhages.

My own experimental investigations, which I report here in connection with those reported by Ederlen and Loeb, and previously mentioned on page 665, were made as follows: the phosphate meal was dissolved in water and filtered through linen, in order to retain larger particles, and the filtrate was injected into the bronchi through the larynx by means of a Pravaz syringe. I thus produced two different processes: a diffuse pneumonic affection principally in the lower lobes and small foci consisting of granulation-cells and without exception containing one or more of the foreign bodies in their center. The mode of origin and significance of these foci have been discussed at length in another place (page 674). The former condition consists in diffuse swelling of the alveolar epithelium and a widening of the alveolar interstices, associated with a uniform and wide-spread exudation of red blood-corpuscles into the alveoli and, in even greater profusion, into the interstitial tissue.

I shall have to postpone my report of this pulmonary inflammation, as at present the number of my cases is too small. But even at this early date I believe I can positively maintain that the effect of inhalations or injections of Thomas phosphate meal on the pulmonary tissue is almost entirely chemical, as otherwise it would not be possible for changes to appear which, according to clinical and experimental demonstration, so materially differ from the effects of the inhalation of other kinds of dust. The reported granulation foci, illustrated on Plate 6, figure 9, in the center of which one or more granules of Thomas phosphate meal are constantly found, are most likely the result of combined mechanical and chemical influences; but the acute pneumonic process cannot possibly be explained in any other manner than by the effect of ingredients of Thomas phosphate meal which dissolve and chemically influence the alveolar epithelium in a detrimental manner.

SYMPTOMS.

The description of the clinical manifestations may be prefaced by the following words of Merkel: "Unfortunately the clinical pictures even in the genuine forms of pneumonokoniosis are not as characteristic as might be expected, and it is therefore a difficult task to draw a clinical picture of all the diseases included in this description. Moreover, the various kinds of dust that are capable of producing the disease possess distinct peculiarities, and the same physician rarely has the opportunity to observe all the various kinds of dust-inhalation. In spite of the very respectable extent of the clinical material at my disposal, I must confess at the outset that I rarely made the diagnosis before the subject was brought to the autopsy table. But the more I study this question, the more firmly I am convinced that in many cases the presence of dust in the lungs is quite harmless, while, on the other hand, it happens more often than is generally supposed that an apparently ordinary disease of the respiratory organs is primarily due to the inhalation of dust; and I believe we shall yet learn to make the diagnosis during life with more certainty than has been possible in the past."

An accurate analysis of the symptoms, like the description of the pathologic changes, demands a strict separation of the different varieties of dust. The inhalation of coal-dust alone will be considered first; next, the inhalation of iron- and stone-dust, both of which are found in varying quantities, either by themselves or in combination with other kinds of dust, in certain industrial establishments; and, lastly, the inhalation of Thomas phosphate meal will be made the subject of a special discussion.

The inhalation of coal-dust is relatively harmless. Although Crocq regards the appearance of anemia in coal-miners as the earliest effect of anthracosis, other conditions, such as the want of light in the coal-mines, the vitiated, stagnating air that the miners breathe, and insufficient food and clothing, are equally, if not more important etiologic factors.

The asthma which appears in these laborers should be of greater importance, as it is undoubtedly a direct consequence of the pulmonary changes. Its appearance is probably due to the combined effect of plentiful inhalation of coal-dust and obstinate bronchial catarrh. In one way, as mentioned, the latter favors the penetration of dust particles into the tissue, as in catarrh a comparatively larger quantity of coal will adhere and cannot be expelled again, as is done by a normal mucous membrane. In the second place, the bronchial catarrh in itself, on account of secretion of mucus in the smaller bronchi, contributes to the restriction of the respiratory surface, and consequently leads to dyspnea, and it is unquestionably true that the inhalation of coal-dust tends to render an existing bronchial catarrh permanent. Therefore coal-dust and bronchial catarrh represent a vicious circle.

The character of the sputum varies according as the catarrh is acute or chronic; but if the miner has followed his avocation for any length of time and too long an interval has not elapsed since he has given it up, the sputum, which usually has a mucoid, glairy appearance, invariably contains pin-point to pin-head-sized particles which on microscopic examination are found, as a rule, to consist of aggregations of large, round cells, with or without nuclei. These cells contain a considerable number of black granules of fairly constant size, and their identity with alveolar epithelium containing coal-dust cannot be mistaken by any one who is in the habit of examining anthracotic lungs under the microscope. The uniform size of the particles is explained by the fact, which has already been sufficiently discussed, that only the smallest particles are carried into the alveoli by the inspiratory air-current and later expectorated.

In spite of the presence of these changes, examination of the lungs need not necessarily yield objective signs. The symptoms of an already existing catarrh are of course independent of the deposition of coal. The same may be assumed of emphysema; it is no more frequent in coal-miners than in other laborers who are forced to inhale foul air and are exposed to all kinds of weather.

If any physical signs are found in the lungs, they almost always consist of the signs of consolidation over one or both upper lobes. Dulness and bronchial respiration are present; and crepitant râles, especially over the pulmonary apices, will rarely be absent.

According to the view which is here adopted, one may conclude that around an originally existing focus in the pulmonary apex, which is probably always of a tuberculous nature, an extensive black induration, or rather, more correctly, a chronic pneumonia, has developed with wholesale deposits of coal-dust. I was able to demonstrate such a finding at the autopsy of a laborer who for a number of years had been employed in the gas-works of this city [Magdeburg].

If, on the other hand, a tuberculous infiltration develops in an individual who for a long time has been exposed to an atmosphere of coal-dust, particles of coal which had been embedded for a long while in the peribronchial tissue must be expelled with the disintegrated pulmonary tissue. Thus it may happen that individuals who have never been much exposed to a coal-dust laden atmosphere, or who have not been exposed for some time and have not had any coal-dust in the sputum, suddenly begin to expectorate black material. If a careful microscopic examination is made, elastic fibers are almost always found in the sputum in such cases.

While, therefore, the inhalation of coal-dust is followed by indirect results,—*i. e.*, increase and longer duration of an already existing catarrh, and chronic inflammatory changes in the neighborhood of already existing focal diseases,—the inhalation of iron- and stone-dust has directly injurious effects upon the pulmonary tissue.

Merkel, based on his ample clinical observations, described the effects of inhalations of iron-dust, which agree with those of stone-dust, as follows:

Regarding the first onset of catarrh, everything is exactly the same as in anthracosis. It is the first symptom; probably no one who exposes himself to an atmosphere filled with iron-dust is exempt. The same is true of the sputa if we substitute iron-dust for coal-dust, and remember that the presence of iron is readily detected by treating the sputa with dilute hydrochloric acid and adding ferrocyanid of potassium; a reaction of Prussian blue removes every doubt.

A variable interval elapses between the first exposure to the dust-laden atmosphere and the appearance of the earliest symptoms of any gravity; it may be only nine months or as long as twenty-five years. The earliest case on record occurred in an individual who was undoubtedly diseased when he began to work at the trade. The average duration of the disease, counting from the first grave symptom that caused the workmen to seek medical advice to the fatal termination, was two years; the general clinical picture and all the symptoms were the same as in ordinary pulmonary phthisis. The cough is at first intermittent and later becomes constant. Expectoration of mucoid and later purulent sputa containing macroscopic particles of dust, physical signs of chronic infiltration, pleural complications, cavity-formation, fever, night-sweats, diarrhea, loss of strength, intercurrent attacks of acute pneumonia, partial emphysema, and bronchiectases make up the clinical picture. The disease may lead to every conceivable consequence, such as congestion in the greater and in the lesser circulation, hydropericardium, hydrothorax, ascites, enlargement of the liver, albuminuria, and anasarca. In such a way sooner or later death ensues, and, except for the sputa, siderosis does not differ in any respect from the usual picture of pulmonary phthisis of the lungs.

Only the inhalation of Thomas phosphate meal leads to acute inflammation of the pulmonary tissue. According to Ehrhardt's report, the cases generally progress like croupous pneumonia. They commence with chills, followed by stitch in the side. The sputa are bloody or rusty. Herpes appears often (6 times in 20 cases). Physical examination shows the characteristic signs of an extensive pulmonary consolidation, mostly affecting one or both lower lobes. Departing from the course of common croupous pneumonia, severe delirium frequently set in during the initial stage and sometimes increased to maniacal restlessness. Most patients also had severe dyspnea, sometimes even orthopnea. Finally, an almost complete cessation of expectoration was observed to be a constant symptom. The disease terminated by crisis in more than half of the recovered cases. The general course was very unfavorable. Six died out of twenty affected. Of great influence for a lethal termination were: age (three of the deceased were respectively thirty-five, forty-five, and fifty-seven years of age), renewed attack (in two), preceding bronchial catarrh (in one case).

Pleural effusions often accompanied the disease. I have already mentioned in connection with the pathology that the disease should not be taken to be croupous pneumonia, but must be considered as atypical pneumonia (see page 579). This view is supported clinically by the early appearance of delirium, the severe dyspnea, the atypical expectoration, and, last but not least, the great mortality.

PROPHYLAXIS AND TREATMENT.

In regard to pneumonokoniosis, prophylaxis has a very difficult task to accomplish. It is easy to define, but difficult to fulfil. It consists in the prevention of the penetration of dust particles in the different trades and factories which are associated with the development of large quantities of dust. As far as the inhalation of dust occurs outside of the dust-producing manufactories, it does not amount to much, and is not injurious to health, consequently it need not be considered.

But the physician in recommending prophylactic measures in the sanitary interest of the laborer has to consider the life of the respective industries, otherwise he might take radical steps and plead that no laborer should expose health and life to so injurious an occupation. This, however, might ruin professions which yield to many men the income which is required for their living.

Legislative steps should be taken to avoid the development of dust as much as possible. For this purpose the moistening, whenever possible, of the dust-producing materials is of advantage. Wherever this cannot be done, it may be possible to settle the dust in the neighborhood of the laborer by sprinkling. Furthermore, the working rooms should be comparatively large and well ventilated, and ought to be provided with a plentiful supply of fresh air which completely removes the dust-filled air.

Such principles are long since acknowledged and carried out as far as possible. Protective means against the inhalation of dust which the laborers should place in front of mouth and nose proved to be either inadequate or inapplicable, as is exhaustively explained by Merkel, who also refers to Härting, Hesse, and Schreiber.

While these precautions should be taken to make the trade as such as safe as possible, individual prophylactic steps should be insisted upon more strenuously than they have been in the past. Laborers must have healthy respiratory organs if they intend to work at a trade which is connected with the development of large quantities of dust. Such a stipulation is humane and should be enforced by law. Everybody who applies for a position in such a trade should have to undergo a careful physical examination. If only the slightest abnormality of the pulmonary apices can be demonstrated, if only the least sign of an emphysema exists, even if it can be proved that several attacks of bronchial catarrh have preceded, the employer must be prohibited from engaging such a man. The same should apply to such laborers who have been working at a dust-producing trade and during their occupation have acquired an affection of the respiratory organs. A certain age limit should also be enforced, as, according to Ehrhardt's observations, people of advanced age are most exposed to danger.

The first therapeutic indication is to remove the workman from the dust-laden atmosphere in which he is working on the first appearance of symptoms pointing to disease of the respiratory apparatus, even though it be nothing more serious than a simple bronchitis. It is quite immaterial whether, in the case of coal-dust, for example, we regard the inhalation as the direct cause of the bronchitis, or adopt the view that it is incapable of producing bronchitis and merely delays recovery from an already existing bronchial catarrh and favors the entrance of dust particles into the pulmonary tissue. Whenever catarrh has actually developed, the inhalation of coal-dust must be avoided as scrupulously as the inhalation of iron-dust or stone-dust, which in themselves suffice to bring on a lesion of the bronchial mucous membrane beginning with catarrhal manifestations.

If the physical examination reveals the existence of a lesion in the pulmonary tissue itself, it makes little difference whether an already existing affection of the apex has become complicated by the development of extensive chronic consolidation in its neighborhood, as usually happens when the individual has been exposed to coal-dust, or whether the focal disease has been produced by the inhalation of particles of iron or stone which, on account of their higher specific gravity, probably exert a more deleterious influence than coal-dust; in most instances the disease is tuberculous in character; *i. e.*, it is associated with the presence and the malign influence of tubercle bacilli, which must be fought with appropriate hygienic and dietetic measures, as well as with special methods of treatment. A detailed description of the latter would be superfluous in this connection.

Acute pneumonia caused by inhalation of Thomas phosphate meal is to be treated according to the principles formulated in the discussion of croupous and atypical pneumonia.

CHRONIC PNEUMONIA.

PATHOGENESIS AND PATHOLOGIC ANATOMY.

THE description which was first given by Laennec comprises the entire sphere of the much-discussed chronic pneumonia. His words are: "Around cavities which originate from a gangrenous eschar of a lung, I at times found the pulmonary substance much harder than in simple hepatization; the tissue grated under the knife. The cut surface was more distinctly granular than in acute pneumonia. On tearing the tissue, the granulations appeared to be more sharply defined, firmer, and drier. They showed the different shades of acute hepatization, but violet-gray and blue-red prevailed, while only a few yellow points were visible. The entire tissue could not properly be called moist; by scratching with a knife a little moisture was obtained."

He also uses the term chronic to describe an acute pneumonia that has been arrested in its course by venesection or some other antiphlogistic procedure which has failed to bring on prompt resolution or prevent an aggravation of the symptoms. Thus he saw pneumonia remain for two months in the stage of engorgement which before the end changed into simple edema. In the small number of cases which he had a chance to dissect he found areas of firmer and less moist consistency than is observed in acute hepatization. In the interstices between these areas the pulmonary tissue appeared much infiltrated by serum which was interspersed with yellow points. This fact, as well as the yellowish color of the pulmonary tissue, seemed to indicate that the resolution of a pneumonia had entered the purulent stage.

This description includes the questions which are still unanswered at present. The question whether chronic pneumonia results from acute genuine pneumonia has never been finally settled to this day, although Stokes positively maintained it to be a fact. "He absolutely concurs with Forbes, that although primary chronic inflammation of the lungs is very rare, the secondary form resulting from an imperfectly dissolved acute pneumonia, or as a complication of other morbid conditions, is not rare."

If we follow Laennec's description of the disease, to begin with, we have to consider the fact, which has been overlooked, that two different kinds of processes have been comprised under the same designation. In one it is a question of purely secondary changes. A gangrenous area, or, according to Laennec, a hemoptysis, or even tuberculous cavities will produce chronic pneumonia of the surrounding pulmonary tissue.

Therefore there are no common genetic relations between cause and effect. The pneumonic affection merely represents a kind of encapsulation of a necrotic portion of lung tissue; it corresponds to hyperostitis surrounding a necrotic portion of bone; according to the structure of the pulmonary tissue, however, the encapsulation is not limited to a narrow field, but under unfavorable conditions the complicating inflammation of the pulmonary tissue may gradually spread toward the periphery. Any pneumonia of this kind may surely be called consecutive.

Entirely different is the course in those cases in which the affection is said to have been caused by acute pneumonia. In this case it is not at all necessary to decide whether this is really the case; but simply to demonstrate that a chronic pneumonia which, according to the opinion of most authors, is to be attributed to acute pneumonia, is really in every respect entitled to a separate position among the above-mentioned consecutive forms. Whether it originates from genuine pneumonia, and therefore has a genetic connection with the latter, or represents an independent disease, under all circumstances this kind of chronic pneumonia must be regarded as primary, since no other change of the pulmonary tissue has preceded.

Primary chronic pneumonia demands greater consideration, partly on account of its independent onset, and partly on account of the more considerable extension of the process from the beginning.

The earliest and best macroscopic description is that of Marchand, and I cannot do better than repeat his words.

If the induration is observed early, it differs in color but little from gray hepatization, especially if an entire lobe is involved. However, the coloring is more or less reddish according to the greater or lesser development and congestion of the vessels; in greater development of the same it may even become dark brownish-red. The extreme resistance, elasticity, and toughness of the parenchyma are characteristic; one tries in vain to penetrate the tissue with the finger, which can easily be done in recent hepatization. In examining the cut surface it appears granulated, but the granulation is much less distinct than in recent hepatization; the impression is created at once that the contents of the alveoli are much more closely connected with the walls, as can easily be demonstrated by scraping with a knife which produces but a small quantity of slightly turbid fluid and no larger granules.

Marchand also emphasizes the occurrence of firm pleural adhesions of long standing in all cases of chronic pneumonia. This leads to better vascularization and then offers better conditions for the new-formation of tissue. Furthermore, these adhesions diminish the activity and elasticity of the lungs, and therefore diminish the access of air.

Von Kahlden lays special stress upon the latter point. The decreased access of air may be followed by mechanically increased blood-supply, and the inaction of the lungs may interfere with the removal of fibrin by means of resorption and expectoration.

Fraenkel does not consider pleuritic adhesion to be of such importance. In the second of the cases reported by him such adhesions of long standing were not present at all. According to my observations as well, they need not necessarily exist in pronounced cases of chronic pneumonia.

A microscopic examination is required to obtain a clear idea of the pathogenesis. "Until the microscopic appearances corresponding to one of the traditional or newly introduced designations have been determined, we can have no accurate knowledge of the respective condition. It is therefore necessary to demonstrate the elementary changes that produce the induration, and from them to develop the physical signs which until now have constituted the whole of our knowledge of the conditions." Thus writes Heschl, who was the first to demonstrate the more minute pathologic changes. His investigations led him to the conclusion that induration of the lungs consists in a new-formation of connective tissue, communicating with the trabecular framework of the lung and permeating the basal tissue of the organ. This new connective tissue he derives from the nuclei of the alveolar capillaries. During the subsequent course of the disease the alveolus may become permanently occluded. This occlusion is probably effected by two processes, in addition to the contraction of fibrinous products deposited in the alveoli. One of these processes consists in the proliferation of the connective tissue which begins in the blood-vessels of the alveolar wall until finally the pulmonary vesicle is completely occluded. The only circumstance that negatives this assumption is the small volume of lungs that have become obsolete; hence the ultimate obliteration of the alveoli probably depends chiefly on another process which, in addition, tends to obliterate the blood-vessels—namely, the shrinking of the masses of newly formed connective tissue in the lung.

An observation of Heschl deserves special notice. It is the presence of capillaries running a slightly tortuous or absolutely straight course and either terminating abruptly in the cavity of the alveolus with or without a terminal arborization, or passing on to the opposite wall and penetrating its tissue. His interpretation of these capillaries, however, is faulty. The entrance of the capillaries into the lumen of the alveoli he believes to be due to a loosening of the normal pulmonary capillaries from their accustomed firm surroundings by the newly formed connective tissue derived from the spindle-shaped elements, and by a simultaneous thickening of the trabeculæ; the capillaries thus become almost or completely isolated for considerable distances.

In the two cases which Eppinger had an opportunity of investigating he found a powerful hyperplasia of the interalveolar connective tissue whence similarly formed fasciculi of spindle-shaped proliferating connective-tissue cells projected into the alveoli like plugs. They contained newly formed vessels. He assumes that the material for the hyperplastic proliferation is derived from an increase of the adventitial nuclei of larger venous trunks and from new elements resembling lymphoid cells or white blood-corpuscles, around narrow vessels and capillaries. He believes the starting-point of these elements to be the pleura, which was greatly thickened in both his cases, and from the inner layers of which processes of hyperplastic connective tissue in all stages of development, from spindle cells to completely formed fibrillar connective tissue, extended into the pulmonary tissue.

Ackermann and Thierfelder demonstrated in the case which they elaborately described, clinically and anatomically, a considerable development of the periarterial and of the interalveolar tissue, and concluded that the alveoli lose in width in proportion as the volume of their septa increases. The constriction and final destruction of the alveoli occurs therefore in such a manner that they are closed by overgrowth from their periphery. The authors also found, not rarely, in some specimens throughout, that a spherical mass of connective tissue with a short pedicle had grown from the interalveolar tissue into the alveolus and filled all but a narrow portion near the periphery. The hyperplasia of the interlobular connective tissue is consequently of not much importance for the alveolar atrophy. Moreover, a considerable development of the alveolar epithelium was found in the remaining alveoli so that the lumen was completely occluded.

According to Marchand, the great mass of the connective tissue concerned in the production of pulmonary induration is found in the alveoli themselves; later it melts down along with the alveolar wall, and ultimately undergoes cicatricial contraction. Most of the alveoli contain club-shaped or irregular masses which are attached to the walls by peduncular processes and by isolated fibers and blood-vessels; the peripheral portion consists principally of densely packed spindle cells, which also extend into the pedicles, while the central portion frequently retains a fairly homogeneous, granular, and fibrillar structure. The development of the connective tissue, he says, begins in the originally fibrinous contents of the alveoli which form the nucleus; the fibrinous mass condenses and then represents a fairly homogeneous, granular, and fibrillar mass, around and through which the young connective-tissue cells proliferate from all directions. It is more than likely that the latter are nothing but converted lymph-corpuscles.

The growth of connective tissue is accompanied *pari passu* by the new-formation of blood-vessels. The latter begins in the vessels of the alveolar wall, as Heschl and Eppinger assumed. Marchand deduces from the microscopic findings that the young blood-vessels are formed from sprouts on the capillary wall; these nucleated sprouts become elongated, and finally undergo canalization. It is at least certain that the formation of spindle cells in the alveolar contents precedes the development of the blood-vessels.

The participation of the interalveolar and interstitial tissue during the onset of the process is of no importance. The principal change consists of cellular infiltration, mostly in the immediate proximity of the thickened pleura and in the larger interstices, also in the adventitia of the larger vessels and bronchi; not until the later stages thickening of the alveolar walls takes place in connection with shrinkage of the connective tissue and atrophy of the alveoli.

"Finally, with regard to the name of this termination of pneumonia it seems proper, in order to avoid misunderstandings, to drop the old designations, induration, interstitial pneumonia, chronic pneumonia, for this condition; partly because each of them is also used for other conditions, partly because it is not expressive of the nature of the process. As it is, a question of the substitution for the fibrinous exudate which fills the alveoli of connective tissue which finally leads to the formation of a dense fibrous mass occupying large areas of the lungs, the entire condition, which originates in fibrinous pneumonia, might be designated chronic fibrous pneumonia."

Von Kahlden investigated five cases of induration of the lungs following croupous pneumonia. Two to five weeks after the onset of the disease they terminated in death. All the cases presented a nearly uniform histologic picture which showed a new-growth of connective tissue taking place almost exclusively within the alveoli, and in which, undoubtedly, the first appearing fibroblasts originated in the connective tissue of the alveolar wall. Apart from a moderate enlargement by ectatic vessels, the alveolar septa showed a slight thickening, depending upon an increase of their cellular elements. The thickening of the alveolar walls does not increase in the later stages, so that the connective-tissue induration of the lungs is almost exclusively the result of neoplastic connective tissue within the alveoli. The vascularization of the newly formed connective tissue is effected by newly formed vessels which extend from the pre-existing vessels of the alveolar wall into the plugs that fill the lumen.

Clark considers fibrous induration of the lungs to be an independent disease, and assumes three possibilities of its origin. According to his view: (1) Without any other affection of the pulmonary parenchyma proper, the pleura may become thickened, send out coarse connective-tissue fibers into the parenchyma, and irritate it to connective-tissue induration. (2) The interlobular tissue may act in the same manner by involving the alveoli in the process without any independent affection being present in the latter. Clark has seen cases in which the principal changes took place in the interlobular tissue, while the interalveolar septa were only thickened by spindle-cell tissue without the lumina of the alveoli having been filled with exudation masses. (3) Exudation into the lumen of the alveolus may take place; but the exudate is altogether different from that which occurs in fibrinous and catarrhal pneumonia. The exudate rapidly becomes converted into fibrous tissue, the spindle cells of which are evidently derived from the small corpuscular elements underneath the epithelial cells in the intervascular spaces and on the walls of the alveoli, but not from the alveolar epithelium proper.

According to Molly Herbig, the most conspicuous objects in microscopic sections are the restiform structures, which are often so long that they are bounded by thirty or more alveoli. The diameter varies slightly in different portions of the structure, which is partly straight and partly curved in various ways. The cords frequently divide into two or more branches, which may again subdivide, so that the entire structure assumes an arborescent appearance. At various points the main trunk and lateral branches present local enlargements which bulge more or less into the surrounding tissue. These enlargements or excrescences occasionally enter the adjacent alveoli and fill them more or less completely, forming club-shaped or plug-like masses. Some alveoli are also found that do not contain these masses. The walls of these alveoli are only slightly thickened, and the lumen usually contains cellular material of varying density.

These arborescent structures that have just been described are undoubtedly found also in some of the bronchi, where they partially or completely fill the lumen. The neoplastic process has its origin in the terminal branches of the bronchi.

The surface of these club-shaped masses and bands of newly formed tissue are frequently covered with a layer of epithelial cells, as Marchand has stated. These cells are either in immediate contact with the connective tissue or separated from it by a cleft-like interval. Within the alveoli, especially on the acuminated portions of the occluding plug, the epithelial cells continue to grow for a certain distance independently, in a single or double row, thus extending further into the lumen, or even as far as the alveolar wall.

Kohn believes that the interlobular connective tissue, under the influence of some irritation, proliferates and sends out processes into the adjacent alveoli; the fibrous exudate here becomes organized and penetrates by way of the pores into the next, and then into the third adjoining alveolus; that is, the interlobular or pleural connective tissue plays the principal part in the process. According to Molly Herbig, this opinion is contradicted by the fact that, as can be observed macroscopically, just under the surface of the pleura a narrow streak of absolutely airless tissue exists in the lungs which is already plainly consolidated, and that, as the microscopic examination will prove, there is no question of plug formation in the alveoli as yet, or that the first bulb-like spores of the new-formed restes just advance into these alveoli. However, Kohn is perfectly right when he maintains that the mentioned connecting cellular rows, which he has described, penetrate the alveolar walls, and considers a basis for this phenomenon the fact that in fibrinous pneumonia the fibrinous plugs also are connected with each other by delicate fibers which pass through the wall, because it is certainly correct that the connective-tissue development of the induration process follows the tracks marked out by the fibrin.

Borrmann, a disciple of Orth, joins the opinion of former investigators, and lately principally that of von Kahlden, that in connective-tissue induration of the lungs we have to deal with a fibrous proliferation in the lungs which originates in the alveoli; a process which is quite analogous to that of thrombus organization from the vascular wall.

But it remains an unanswered question for the present whether the first-appearing fibroblasts originate in the endothelia of the vessels contained in the interalveolar septa, as is assumed by Fiegler and others, or from the connective-tissue cells of the parenchyma, as is maintained by Orth and others, or whether they have still another source.

This apparently quite exhaustive compilation only comprises a part of the publications in reference to chronic pneumonia. As the papers of Marchand, Amburger, and a condensed, very able report of von Kahlden contain a complete bibliography, it may be admissible to specially mention here only those papers to which confirmatory or supplementary reference had to be made in communicating my own observations.

A microtome section from a lung in the stage of full development of chronic pneumonia offers a truly striking picture after proper staining; especially if the Biondi-Heidenhain triple stain (see page 396) be used. At numerous places single parts of the section comprising ten and more alveoli appear to be uniformly changed. The alveoli are slightly diminished in size throughout and filled by a globular mass which on a fibrous basis contains a fairly large number of nuclei. Within this newly formed mass, usually near the center, are capillary vessels. As the individual red blood-corpuscles, when stained with the Biondi-Heidenhain mixture, take a golden yellow color and almost always completely fill the blood-vessels, forming a compact column, it is easy to see that the corpuscles and the wall of the vessel which contains them are in direct communication with the capillary vessels of the alveolar wall (see Plate 4, Fig. 11). The capillaries found within the alveolar contents must therefore have grown directly from the normal capillaries of the alveolar wall. In one instance I even saw a capillary vessel that passed through the fibrous contents of two alveoli, so that it must have passed through the alveolar wall.

Some alveoli are not filled with fibrous tissue, but with a hyaline mass which has no vessels. This turns rosy on applying the above stain. In rare cases one-half of the alveolar contents consists of this hyaline mass, the other of fibrous vascular tissue.

But this fibrous tissue, where it appears alone or with the hyaline mass, does not always completely fill the alveolus, and such places offer conditions which can be utilized for the explanation of the entire process.

The central part of the alveolar contents contains a capillary vessel, sometimes two. The vessel is surrounded by numerous round cells; outside of these are oblong cells with oblong nuclei. At both ends of these cells are processes, sometimes of considerable length. At different places the cells are transformed into long fibers because their nuclei have disappeared. Such findings speak very forcibly for the opinion which I expressed long ago,[5] that the connective-tissue fibrils emanate immediately from the cellular protoplasm; here it will be sufficient to prove the fact that a new formation of connective tissue takes place.

Between this unquestionably newly formed mass—which consists inside of round cells with capillaries, outside of spindle cells with fibrillary processes and connective-tissue fibrils—and the alveolar wall remains an empty space, as stated, but always in the shape of a larger or smaller crescent; for the entire new-formation is attached to some place of the alveolar wall in the same manner as a bud to its stem. It is at such a place that the capillary vessels enter the newly formed mass from a normal alveolar vessel in the alveolar wall. In the remaining empty space between the new-formation and the alveolar wall are almost without exception large cells with a wide protoplasmic margin which can only correspond to swollen alveolar epithelia. To consider them to be the new-formed epithelial cover of the bud- or button-like fibrous mass which advances into the alveoli is not admissible, as they never lie on the new-formation, but are either free in the empty space or on the alveolar wall.

It is just as important for the elucidation of this process that at numerous places several, even many, of these bud-like growths of the alveolar spaces are connected with each other by bridges which pass through the alveolar walls. These bridges are composed of long-drawn-out, nucleated, spindle cells.

But nowhere an active participation of the alveolar walls in the entire process can be demonstrated. Nowhere exists an increase of cells or a broadening of the interstices. That the process in the alveolar walls is entirely passive I am inclined to conclude from the following facts: Sometimes there are found newly formed nodes of tissue of the described kind, *i. e.*, alveolar new-formations consisting in capillary vessels, round cells, spindle cells, and connective-tissue fibrils, of so large a volume that it must be presumed they completely fill an infundibulum; therefore, they must have completely obliterated the alveolar wall. Then, also, the connecting bridges between the contents of neighboring alveoli are often so wide that here also an obliteration of the interalveolar tissue must have taken place. That such an obliteration takes place has also been presumed by Aldinger. These are the findings which are valuable for the explanation of the entire process.

As there cannot be any doubt that newly formed tissue exists within the alveoli, the next question will be: From which elements do these new-formations originate? A participation of the alveolar epithelium is to be excluded. In cases in which the alveoli are not completely filled by the new-formations, where therefore an empty space exists, the alveolar epithelium is seen to be intact, although swollen. Therefore it was necessary to return to the remaining connective-tissue elements of the pulmonary tissue for their origin. On this theory, Kohn attributes the process to the fact that the interlobular and even the pleural connective tissue sends out sprouts into the alveoli. Molly Herbig correctly objected to this, as the pulmonary tissue immediately under the pleura often appears fully intact.

However, Kohn's view that in chronic pneumonia the newly formed tissue follows the same lines as does the fibrin exudation in acute croupous pneumonia is correct. The fibrin of neighboring alveoli is connected by communicating fibers which penetrate the alveolar wall. How this occurs has been described in detail on page 398. There I explained the passage of the fibrin threads through the alveolar wall by the fact that fibrin originates in the blood exclusively, and for this reason must principally be found in the neighborhood of the capillaries. As the capillaries penetrate the alveolar wall, the fibrin is bound to accompany them everywhere and is, therefore, present in the wall of the alveoli as well as in the lumen of the same. The strongest fibrin threads must even be in the wall, because the fibrin cannot spread out as easily here as in the alveolar lumen (see figure 24, page 398, and Fig. 2 on Plate 4). This explanation does away with the supposition of special stomata in the alveolar wall.

Only one difference exists between the communicating bridges which penetrate the alveolar walls in chronic pneumonia and the fibrin threads of acute pneumonia. In the former they are generally materially stronger. Therefore at first a pressing apart of the tissue elements of the alveolar wall must take place and then an obliteration of the same must occur. This fact was not considered by Molly Herbig when he assumed that the entire process originated in the final terminations of the bronchi, because on such a supposition the passage through the alveolar wall cannot possibly be explained.

When I myself went over a large collection of preparations which plainly brought to view the tissue-buds in the alveoli connected by communicating bridges, I was involuntarily reminded of microscopic pictures which I had seen in my experimental investigations of the healing of wounds. I was able to demonstrate in my work on the origin of the connective tissue[5] in muscle wounds, which I caused to heal subcutaneously by displacing the skin over them, that into the empty space which is produced by retraction of the muscle fibrin enters first, that white blood-corpuscles enter into this fibrin, and that from these the connective tissue creating the scar is formed.

In spite of many contradictions, I think I am entitled still to uphold my view at present that the connective tissue emanates from white blood-corpuscles in the manner I described at the place referred to.

The observations made in chronic pneumonia merely tend to confirm this view of mine. The theories in regard to the origin of the connective-tissue formations within the alveoli, which have been mentioned so far and which do not appear plausible, collapse as soon as we cease to rebel against the fact that white blood-cells are the ancestors of the long-drawn-out spindle cells observed in these formations. The explanation of the whole process immediately becomes quite simple. According to general agreement, we may consider to be the first change the presence of fibrin in the alveoli which, as in acute croupous pneumonia, also penetrates the alveolar walls and connects the contents of several alveoli. This is followed by the escape of white blood-corpuscles, which, just as in wounds, embed themselves in the fibrin. Then nothing is more obvious, especially in view of the absence of all necrobiotic changes in the white blood-corpuscles, than the presumption that the white blood-corpuscles which have entered the fibrin undergo progressive changes, and, in just such a manner as I have described in connection with the genesis of connective tissue, are partly used for the construction of the vessels which are present in the center of the alveolar formations, and partly undergo a transformation into spindle-shaped cells and connective tissue.

On this basis the relations of chronic pneumonia to acute croupous pneumonia are placed in a different light. A number of weighty reasons speak for the supposition that the two diseases are in very close genetic relationship. To begin with, Fraenkel, in one of his cases, found Diplococcus pneumoniæ just as in croupous pneumonia. Further, the clinical onset of the disease, at least in some cases, is the same as in croupous pneumonia. Finally, the pathologic change in the beginning is the same, consisting in a swelling of the alveolar epithelium and an exudation of fibrin into the alveoli.

Only in one respect I have to maintain a view which deviates from the current explanations of the genesis of the disease. Chronic pneumonia does not correspond to croupous pneumonia in its characteristic stages; its course corresponds only with the first stage of croupous pneumonia, *i. e.*, swelling of the alveolar epithelium and exudation of fibrin, but from then on it proceeds in a different manner. A hemorrhage into the alveoli, which in croupous pneumonia follows the exudation of fibrin, does not occur; therefore the white blood-corpuscles cannot get into the alveoli which were previously filled with red blood-corpuscles; they enter the fibrin, but much more slowly than in acute croupous pneumonia. With this slower process the possibility of organization into connective tissue is connected.

The final termination of the disease consists in contraction of the pulmonary tissue. Wherever chronic pneumonia has its course as such alone without complications, the histologic findings show that bronchiectases cannot possibly form. The bronchi undergo a gradual narrowing, the thorax falls in, even the large vascular trunks are so constricted that the principal branch of the pulmonary artery on the affected side in a manner becomes an aneurysmal appendage to the main trunk, and accordingly causes sounds such as I described in one case.[8] In the latter, the right ventricle was also much hypertrophied. Eppinger and Ackermann also mention the occurrence of hypertrophy of the right heart. The microscopic investigation of the final stage, cirrhosis, shows that the tissue consists of almost nothing but long, broad, vitreous, or hyaline bands. In half a pulmonary lobe, even in an entire one, all the structural parts of the pulmonary parenchyma may be completely obliterated. Only a few small, narrow cell-nuclei are visible in this basic tissue, and more or less considerable accumulations of coal particles bring some change into the monotonous picture of fibrinoid degeneration, the general character of which has been more minutely investigated by Neumann. I shall refer to it again in the paragraph dealing with thrombosis and embolism.

In view of these hyaline bands of tissue, which extend without a break over considerable distances, but one explanation of the occurrence of this alteration is possible. The only structures that correspond in length and direction with these hyaline bands are the masses of fibrin which escape from the blood during the beginning of the disease and infiltrate a considerable number of alveoli; these fibrin masses furnish a foundation for the white blood-cells which emigrate later and become converted into connective tissue. The newly formed tracts of connective tissue, which have gradually caused the atrophy of all the alveolar walls, eventually themselves become obliterated and fall a prey to fibrinoid degeneration.

Changes in the lungs which are known under the name cardiopulmonary defects, and which are dependent upon engorgements in the lesser circulation, complicated the pathologic-anatomic process which has been described so far, because the final outcome of these engorgements is chronic pneumonia, which in comparison to other forms only occupies an etiologic relationship, but pathologically and anatomically is to be considered an analogous process. Hasse was the first to report that he had observed the brown induration of the lungs, which was described by Andral, as a consequence of considerable hypertrophy of the heart. It appeared as if the chronic inflammatory process in these cases had developed from a simple congestive condition, which was kept up partly by the congestion in the lungs, which is always increased in heart disease, and partly by the uninterrupted action of the pathologically increased cardiac impulse; and had increased to a true inflammation. Always the lower lobe, principally the left, was attacked. The parenchyma appeared solid, nowhere permeable or crepitant, although not engorged with blood. The cut surface was smooth and even. Virchow has described this condition as brown induration. Upon opening the thorax the lungs are very prominent, they do not collapse on the admittance of outside air, are more compact to the touch, heavier, inelastic, only slightly crepitant, and often show a peculiar yellowish, brownish, or red-brown hue.

Bamberger calls this brown or pigment induration a very common condition of the lungs in valvular defects, and ascribes it to a hypertrophy of the pulmonary connective tissue with a plentiful deposition of pigment which causes the extraordinary consolidation and firmness of the tissue.

Buhl, later, reported a very remarkable change in brown induration—namely, the finding of high-graded dilatation and elongation of the capillary vessels of the alveoli; this has been confirmed by

Virchow. Feuker, however, maintains[226] that such findings are doubtful, or at least a rare occurrence. He ascribes the induration to the accumulation of cells in the alveoli which, on account of the almost always existing chronic bronchial catarrh, have been aspirated from the finest bronchioles into the alveoli. Contrary to this, Colberg again confirms the findings of Buhl. In six cases of brown induration he demonstrated them twice. Rindfleisch also emphasizes this condition of the capillaries, although he does not deny a certain increase of the interstitial connective tissue.

Orth names an occurrence which has not been mentioned by anybody else in brown induration. He found in the lungs of a woman who died of mitral insufficiency and stenosis, not only numerous capillaries, but also larger vessels (veins), filled with a brown granular mass, and concluded therefrom that a direct pigment formation by red corpuscles may take place without the mediation of cells.

According to my observations, I can confirm the extraordinary dilatation of the alveolar capillaries, and maintain that they take place only in protracted engorgements in the lesser circulation. I have not found that the interalveolar or the interlobular tissue undergoes thickening. But the dilatation cannot be a sign of brown induration, although its presence alone may be sufficient to produce a conspicuous change in the pulmonary consistency. The distention of the capillaries does not occur in every instance, and when it is present, it need not necessarily exist alone. At least I found, in two cases which I investigated for this purpose, other changes which prove that the alveoli participate in the process. In addition to swollen epithelium, not a few red blood-corpuscles appear, a finding which really is to be expected in view of the so-called cardiac-lesion cells being present in the sputum; because, as will be more fully explained in the description of the symptoms, the cells contain yellow and brown pigment granules, and these can have resulted only from the disintegration of red blood-corpuscles. This disintegration, however, must necessarily take place within the alveoli if the alveolar epithelia are to take up the blood pigments.

If, in addition to the swelling of the alveolar epithelium, and to the exudation of red blood-corpuscles, an emigration of leucocytes takes place, as happens in isolated portions of the lung, the alveoli become filled with a cellular material, to the complete exclusion of the air. But even these changes do not represent the final outcome of the morbid process, so far as the alveoli are concerned; if the disease lasts long enough, it leads to chronic pneumonia, with or without dilatation of the vessels, as I am prepared to show by the following observation:

A young man, nineteen years of age, acquired heart disease in 1893, in addition to acute rheumatoid arthritis, and since then, suffered principally from asthma and palpitation, but was able to do office work. Early in March, 1898, he was attacked by bilateral serous pleuritis, which, on March 27th, caused his admittance to the hospital. He was discharged on May 14th, after he had completely recovered from the pleural effusion. But on

June 10, 1898, he had to be readmitted on account of severe increase of his heart disease, and on June 16th he died with all the symptoms of cardiac insufficiency. The autopsy took place twenty-four hours later. As it showed remarkable changes only in the lungs and the heart, these alone will be described here.

The right lung in general is heavy, the apex very emphysematous, the pleura but delicate; in a few places connective-tissue consolidations of long standing are present, but nowhere pleural adhesions. Through the pleura some pale bluish livid spots can be seen, which are very hard to the touch; the pulmonary tissue between these spots is normal, but in an emphysematous condition. The pale livid spots occupy the upper lobe principally; the largest is about as large as a child's fist. An almost equally large spot is in the middle lobe and another one in the upper part of the lower lobe. These spots are completely airless on the cut surface, which is smooth; they are pale gray in appearance; on lateral pressure the finer bronchi discharge but a small quantity of frothy fluid. The left lung, almost nowhere adherent to the thoracic wall, also contains a very emphysematous area half the size of a walnut at the apex; otherwise the upper lobe, especially its upper half, is hard; the tissue shines livid through the pleura. The tissue on the cut surface is completely airless, salmon-colored, with some spot-like, blood-red sprinklings. The lower lobe in general is also somewhat hard, containing little air.

The heart is completely adherent to the pericardium; the muscular structure of the right ventricle is fairly hard, perhaps slightly thinner than normal; in some places the pericardium is here thickened up to 9 mm. A large blood-coagulum is found in the cavity of the right ventricle. The ventricle itself is very wide, the pulmonary valves are delicate. The muscular structure of the left ventricle is brownish-red, not thicker than normal, its cavity is very wide. The pericardium over it is thickened up to 1 cm. by gelatinous swelling of the tissue; the mitral valve is slightly thickened, but otherwise without abnormality. But the auriculo-ventricular opening is very wide; it admits three fingers. On the valves of the aorta, corresponding to the base of the valve, verrucose excrescences of considerable extent are present.

The microscopic examination yielded the following results: A part of the alveoli are here and there completely filled with swollen epithelium, between which plenty of red blood-corpuscles are found; some of the alveoli, however, only contain red blood-corpuscles. In other parts, which are greatest in extent, the alveolar contents are mostly formed by fibrin masses which represent a dense whorl of very fine fibers, but partly also a very delicate small-meshed net. Everywhere alveolar epithelium, small round cells, often also red blood-corpuscles, are embedded in these fibrin masses. But the alveolar epithelia are also found in large numbers within the empty spaces which exist between the alveolar wall and the contents.

In this case, therefore, a process had taken place in the alveoli exclusively, which without hesitation may be designated as chronic pneumonia. This is indicated, above all, by the finding of fibrin plugs containing swollen alveolar epithelium and white blood-corpuscles. Further advanced changes—*i. e.*, new-formations of vascular and connective-tissue—could not be demonstrated within the alveoli.

Whether they do not develop at all in this chronic pneumonia which is caused by engorgement, or whether they do not form until a later stage—these questions I must leave undecided.

ETIOLOGY.

In agreement with the pathogenesis of the morbid process, the etiologic factors must be divided into three groups, according as they are capable of causing—

1. Consecutive chronic pneumonia.
2. Primary chronic pneumonia.
3. Persistent and chronic engorgements in the lesser circulation and resultant changes in the pulmonary tissue.

To the first group belong all focal diseases of the lungs, whether they have their seat in the pulmonary apices and are due to tuberculosis or whether they set in as a consequence of other necrobiotic processes. Thus, among others, the cirrhosis of the lungs which Buhl has described as one of the terminations of desquamative pneumonia should be considered a form of consecutive chronic pneumonia; the same is the case with the black induration of large portions of the upper lobes or of the entire upper lobes, with cavities in the pulmonary apices, which was described under anthracosis.

Some of the pulmonary changes which occur in connection with affections of the esophagus should be included in this group; for instance, the case of Wagner, in which the disease complicated a stricture of the lower esophagus, and the case of Fraenkel, in which it complicated a traction diverticulum.

The presence of an abundance of coal pigment in the chronically inflamed parts of the pulmonary tissue of persons who had not worked in an atmosphere of coal-dust, is explained by the fact that most of the coal-dust particles which are at all suspended in the inspired air and inhaled remain, because only a small part of them can be expelled again owing to the chronic catarrh of the corresponding bronchi.

A great variety of causes may be instrumental in the production of primary lobar chronic pneumonia. If the explanation I have given of the nature of the disease is correct, Laennec's assumption that the disease may be caused by copious bleeding is perfectly justifiable. As, according to the above-mentioned explanation, the process practically corresponds only to the first stage of croupous pneumonia, and the exudation of fibrin caused by the alteration of the alveolar epithelium is not followed by red hepatization, which, as I have shown above (page 401), depends solely on the escape of blood into the alveoli, it is readily comprehensible that copious bleeding, such as was formerly practised, by greatly depleting the vascular system may wholly or partially prevent the development of red hepatization. The deposition of fibrin in the alveoli may then be followed by the other changes which characterize chronic pneumonia.

Intense or long-continued diminution of temperature may have a similar effect. Marchand says that people who, with a beginning pneumonia, sleep in the open, where they are apt to be found dead, expose themselves to a permanent diminution of temperature, and the frequency of induration in these cases may, to a great extent, be due to this fact. In his first case the patient, who had had a thorough drenching at the outset, was treated with frequently repeated baths; the lung was already in an abnormal condition on account of already existing pleuritic adhesions of the old induration in the apex; in the course of the disease finally a pericardial effusion set in. He leaves it undecided whether the treatment with cold baths, which Jürgensen so ardently advocates, was not in this case, with the existing predisposition, very apt to promote the unfavorable termination.

When I was using subcutaneous injections of quinin in doses of ½ gram (8 grains) (see page 540) in the treatment of croupous pneumonia, as I did for several years, it happened once that in a case which was thus treated within twenty-four hours after the onset the pneumonia, although it ultimately ran a strikingly slow course, terminated in complete recovery.

Still, those cases in which injurious influences make themselves felt after the onset of the pneumonia, and cause the termination in chronic pneumonia, are considerably less numerous than those in which a certain disposition to a protracted course exists before the appearance of the disease. Thus, Heschl generally found chronic pneumonia in debilitated individuals, in whom signs of different diseases could be demonstrated, principally intermittent fever of long duration, enlargement of the spleen or liver, dropsy, and degeneration of the kidneys.

Furthermore, according to Marchand, pneumonias which have run their course, and have not undergone complete resolution, but have left a more or less extensive induration with pleural adhesions,—a condition which can be borne without harm if not too extensive,—furnish an important disposition for a subsequent fresh attack of croupous pneumonia. Eppinger ascribes particular importance for the occurrence of chronic pneumonia to preceding hyperplastic processes in the pleura. He assumes that they spread to the pulmonary tissue and cause processes of a like character there.

Marchand also emphasizes that in almost all cases of so-called chronic induration of the lungs extensive callous pleural adhesions are mentioned, and that he did not miss them in his own cases. The injurious predisposing elements of the pleural adhesions are, according to his opinion, diminished dilatability of the thorax and increased blood-supply. For the formation of pleuritic adhesions is accompanied by the development of a great number of collateral vascular tracts which establish a connection between the pulmonary artery and the arteries of the body.

The similarity of the causal conditions in chronic pneumonia appearing in the train of diseases of the heart lies in the engorgement of blood in the pulmonary venous system induced by insufficient activity of the myocardium. Not only valvular lesions, but also chronic myocarditis, and adhesive pericarditis perhaps most of all, may bring about this pulmonary change. An observation of Colberg proves that it is not so much a question of heart disease, as of engorgement of the blood. He found in both lungs, without any valvular affection of the heart, high-graded capillary dilatation and an accumulation of pigmented epithelial cells in the alveoli, principally at the periphery of the lungs. During life brown pigmented sputa had been observed repeatedly, and therefore, in spite of the absence of heart murmurs, a valvular defect had been presumed. At the autopsy this was not found, but, instead, a fairly large development of callous connective tissue at the bases of both lungs in the neighborhood of the large vessels and bronchi, accompanying the latter for a short space. No increase of connective tissue could be demonstrated around the finer bronchi or in the peripheral parts of the lungs. Not the least cirrhotic contraction was present; the lungs were rather bloated, as in patients suffering from heart disease, they did not retract on opening of the thorax, and showed intense black and brown pigmentation. Microscopically, the peripheral parts of the lungs were also in the same conditions as in heart disease. The cause of the increase of connective tissue could not be determined. Colberg correctly assumes that later, when contraction has taken place, the newly formed fibrous connective tissue impedes the circulation in the pulmonary vessels more and more, so that ultimately by stagnation of the blood in the peripheral portion of the lung, which is not cirrhotic, the same changes are produced as if the damming back of the blood had been caused by stenosis of the left pulmonary opening (*ostium venosum sinistrum*).

SYMPTOMS.

The symptoms brought about by *consecutive* chronic pneumonia are best described in connection with the primary affection; they have been fully treated in this volume in the discussion of desquamative pneumonia and of pneumonokoniosis. In the present article we are concerned chiefly with the symptoms of *primary* chronic pneumonia. Wagner says there were, except his own observation, but three cases which enabled him to draw a clinical picture of the disease: Heschl's case, of a boy thirteen years of age; Eppinger's, of a man forty-seven years of age; and Marchand's, of a man twenty-three years of age. According to these reports, the affection was preceded, once or oftener, by severe acute or chronic, bronchial or pulmonary affections. Generally, after a long prodromal stage with symptoms pointing to the lungs, an acute exacerbation sets in which is very similar to an ordinary pneumonia, being characterized by chills, pain in the chest, cough, expectoration, dyspnea, high fever, and feeling of severe illness. At once or in a few days the affection becomes localized in a lobe of the lungs. The results of auscultation and percussion are similar to those obtained in pneumonia; but during the next days the intensity of the dulness and the character of the respiratory sounds change in such a way as not to correspond to croupous pneumonia. The disease remains limited to the one lobe or spreads further during the next days. The dyspnea is not in keeping with the objective symptoms of infiltration, but much more intense; the same is true of the cyanosis. The temperature remains high, or sinks rapidly or slowly, without subjective relief.

The change from lobar fibrinous pneumonia to chronic pneumonia is, according to Fraenkel, characterized by three symptoms: (1) Continuation of fever; (2) persistence of dulness; (3) gradually developing retraction of the pulmonary tissue. The continuance of the fever or its reappearance after momentary subsidence suggests meta-pneumonic empyema, or abscess, or transformation into induration. In the latter case the fever is atypical and may last for weeks, even for over a month. If recovery sets in, defervescence takes place slowly.

I do not agree with Fraenkel in considering the fever to be such a sure criterion. In another place,[13] under the title, "Pneumonia with empyema and abscess of the brain; after two months of good health, lethal purulent meningitis," I have described the case of a merchant, thirty-eight years of age, who, healthy until then, was admitted to the hospital on March 28, 1897. The demonstrated pneumonia of the left lower lobe was complicated by an empyema, while the pneumonia advanced to the left upper lobe. Two weeks after the onset of the pneumonia the empyema was emptied by resection, and with this the fever ceased. Not until two months later fever set in again which was caused by purulent meningitis. At the autopsy, however, besides an abscess of the brain and purulent meningitis, chronic pneumonia was found with complete obliteration of the pleural cavity. Exhaustive microscopic examination of the upper lobe showed all the signs of chronic pneumonia, especially within the alveoli numerous vascular connective-tissue plugs, many of which were connected through the alveolar walls by means of cellular cords.

But for a part of the cases Fraenkel's statement of the course of the fever is undoubtedly correct. I myself had an opportunity of observing what I may call a genuine case of chronic pneumonia from the first onset of the affection until the lethal termination, and of holding an autopsy. The conduct of the fever as well as the other clinical course and the anatomic findings justify an explicit description.

On July 7, 1898, the laborer Adolf D., forty-six years of age, was admitted to the hospital. He stated that he had always been healthy; only during the last four weeks he felt rheumatic pains in the legs, which, however, did not prevent him from working. For two weeks he has had, when drawing a deep breath, slight pains in the chest, in the sacrum, and slight cough. All these molestations increased during the last days. He was much addicted to alcohol; he took 60 pfennig worth—*i. e.*, one liter—of 25% to 30% alcohol per day. He was not infected with syphilis.

Condition on July 5, 1898: Robust man in fairly good condition, with congested face. The tongue is moist, slightly coated; trembles when shown. Strong tremor of the hands; the lower extremities are normal. Patellar reflexes pronounced. The abdomen is soft, not sensitive to pressure; liver and spleen are of normal size. The cardiac dulness extends upward to the upper edge of the fourth rib, to the right as far as the right edge of the sternum; the cardiac sounds are faint, but pure. The action of the heart is frequent, 120 per minute. No abnormality exists over the thorax except over the right fossa supraspinata, where the percussion sound is a little dull; close to the spine slight bronchial inspiration and expiration can be heard. The temperature is raised; the urine, when boiled and nitric acid added, shows slight cloudiness; contains, however, no casts.

July 7, 1898: In spite of having taken 3 gm. (45 grains) of chloral hydrate in the evening, the patient was very restless and had to be isolated. The dulness over the right fossa supraspinata is more pronounced; there exists slight bronchial respiration. In the right interscapular region the percussion sound is dull also, and the respiratory sounds are weakened. The cough is very slight, no expectorations; therefore an investigation for tubercle bacilli cannot be made.

July 9th: Patient is delirious. The face is slightly cyanotic. Incontinence of stools and urine, fever is permanent, pulse frequent.

July 13th: The patient talks incoherently to himself, but answers correctly when spoken to. He says he feels very weak. Continuous spasms of the muscles of the face and of the hands, stiffness of the neck. No expectoration. Over the right apex anteriorly, up to the second rib, the percussion sound is tympanitic and high; catarrhal râles can be heard there; on the right, posteriorly, from the apex to the base, dulness, slight bronchial respiration, and increased pectoral fremitus.

July 19th: Tremor of muscles of hands and face continues. The stiffness of the neck has disappeared; sensorium clear; incontinence of feces and urine. The temperature remains high, as will be observed on the chart (Fig. 32). Condition of the lungs unchanged. Sufficient liquid nourishment and stimulants are taken without opposition almost mechanically. This condition remains continually unchanged, only over the dependent parts of both lungs catarrhal râles can be heard. On August 9th death occurs.

The autopsy takes place eighteen hours after death. The body is in postmortem rigidity, the skin dry, in the dependent parts discolored.

The roof of the skull is heavy, the diploë small, the pia edematous. On taking out the brain a quantity of transparent yellow fluid is discharged; the vessels at the base of the brain are very atheromatous and tortuous. The brain-substance is moderately firm on the cut surface, numerous punctiform hemorrhages appear on the same. A large quantity of transparent yellow fluid is found in the ventricles.

The muscular structure of the body is but little developed, the cushions of fat are abundant. In the pericardium is a moderate quantity of transparent yellow fluid.

The heart shows numerous tendinous areas; the muscular structure is pale yellow and flabby, the edges of the mitral valve are slightly thickened; in the first part of the aorta some calcareous deposits are found.

The right lung is very coarse in the lower lobe and shows increased congestion. The middle and upper lobes are very edematous, the latter conspicuously firm and hard. The left lung at the apex is adherent to the thorax; the lower lobe is edematous, but it is very hard to the touch. The apex of the upper lobe contains a calcareous area the size of a hazelnut.

The spleen is of moderate size, its capsule of gray-brown color; the tissue on section seems brittle, but otherwise does not show any peculiarities. The left kidney is embedded in a rather plentiful adipose capsule, which can be drawn off easily, the same as the connective-tissue capsule. The tissue is cloudy on the cut surface; the design of the straight and curved ducts cannot be distinguished plainly. The right kidney shows the same conditions as the left.

The liver is moderately large, rose-colored, with impressions of the ribs on the surface, on the cut surface of cloudy appearance, the acinus design blurred. The mucous membrane of the stomach is reddened and swollen. No abnormality in the intestine, except a few dark red injected parts are noted.

Single portions of the right upper lobe, of the right and left lower lobe, were properly prepared for microscopic examination. In the right upper lobe the epithelium of a considerable number of alveoli was swollen. The latter were well filled with cells containing no nuclei, and nucleated epithelial cells. The nuclei were but slightly stained blue. The cells were always embedded in a graceful fine-woven net of fibrin threads which intersected each other in all directions. The capillaries of these alveoli were very congested. At other places the fibrin threads of several alveoli were continuously connected and inclosed many round cells besides cells without nuclei.

In the right lower lobe some alveoli contained fibrin exclusively, without any

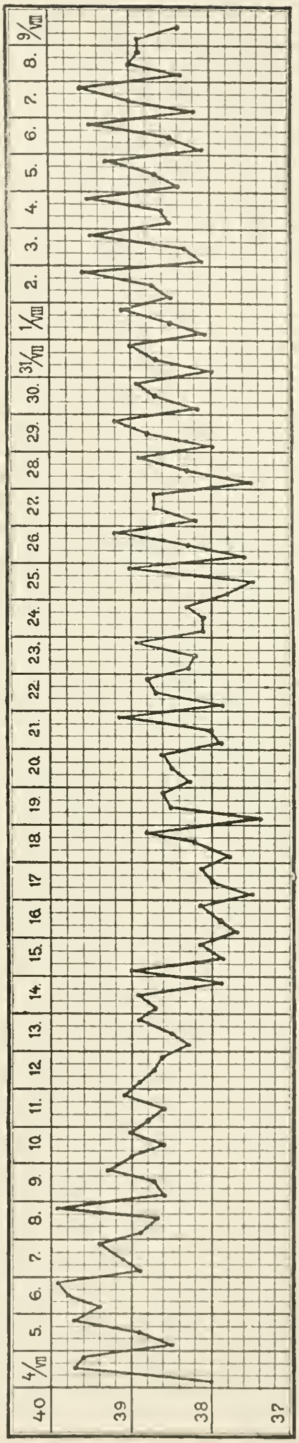

Fig. 32.

cells; other alveoli, however, a few fibrin threads only, interspersed with swollen alveolar epithelium and a few red blood-corpuscles. Here and there the alveoli contained rows of spindle-shaped cells, but were not completely filled.

In the left lower lobe most of the alveoli contained round cells, between which only very few fibrin threads could be detected. In numerous alveoli a larger quantity of fibrin was also found which was arranged in the form of nets and threads. Within the fibrin were small, cellular, multinucleated structures, which evidently originated in white blood-corpuscles. Only in a few places did the alveoli contain nothing but fibrinous masses. The capillaries were filled with pale red blood-corpuscles, but the latter were no longer very well defined.

As mentioned before, this case is remarkable for the high fever which was present from the onset. Then, in an etiologic relation, the abnormally large consumption of alcohol is to be considered, which has also been kept in view by other authors, especially by Josephson in one of his cases. In a clinical relation the absence of any kind of expectoration is remarkable. Therefore an investigation for tubercle bacilli could not be made. The diagnosis of chronic pneumonia was only made possible by the high fever which was present from the onset, by the restriction of the auscultatory signs at first to the posterior right upper lobe, and by percussion signs which in their entirety spoke against a complete consolidation and against a disintegrative process of the pulmonary tissue. That chronic pneumonia had in the mean while attacked also the lower lobe was actually demonstrated only at the autopsy.

The sputum in this disease deserves special mention, principally on account of the admixture of blood. In Eppinger's case it was present during the entire course of the disease; in another case, that of a man fifty-two years of age, which I had observed for over two years, and in which I finally made the autopsy, the sputum contained blood only every few days. It was mixed with mucoid masses and of light red appearance, but it was never expectorated pure, and therefore it could not take the shape of coins in the cuspidor, as in phthisis. Regarding the origin of the blood, I join the opinion of Eppinger and Marchand; undoubtedly, it is derived from the vascular plugs of connective tissue in the alveoli.

In the case just mentioned the sputa were peculiar, which may possibly be characteristic of such a chronic course in general. They were mucopurulent and were expectorated in quantities principally in the morning. In a cylinder-shaped vessel which was filled with water they floated on the surface almost entirely and did not appear arranged in layers as in bronchiectasis.

During the later stages moderate dulness on percussion is present over the diseased portion of the lung. The sound may vary over the same area and become somewhat clearer at different times. On auscultation, the breathing is never bronchial, or at most faintly bronchial; the respiratory murmur is usually weakened. Crepitant râles or small, moist râles are also present; but large, bubbling râles were not present at any time in the two cases which I kept under careful clinical observation.

In chronic inflammatory processes following congestion in the lesser circulation the symptoms are different. The disease develops very gradually, beginning usually as a bronchial catarrh which must be ascribed to the same cause, the stagnation of the blood. Two important factors that influence in a given case the alteration of the alveolar capillaries, and of the alveolar epithelium and the subsequent exudation, are the duration of the process producing the congestion, which is greatest in the presence of cardiac lesions and of pericardial adhesions, and the youthful age of the individuals affected.

An important symptom is the appearance of "cardiac-lesion cells." They have already been declared by Virchow to be transformed alveolar epithelia which contain one or several yellow, yellowish-red, or brownish-red granules of varying sizes. He also refers to their occurrence in cardiac affections, especially in mitral stenosis, and considers the appearance of these cells as a consequence of the damming back of the blood into the pulmonary veins. Friedreich also clearly explains that the mechanical engorgement and distention of the pulmonary vessels, which are brought about by valvular affections, predispose to an edematous infiltration of the pulmonary tissue which sometimes develops slowly, at other times very acutely, and to capillary and larger hemorrhages into the pulmonary tissue. Thus it happens that the hematin liberated by the capillary extravasations, which take place repeatedly and affect extensive areas of the lungs, enters the cells of the alveolar epithelium, where it becomes inspissated and forms yellow, brown, or black granules; the alveolar cells thus become converted into pigment-cells and give the pulmonary parenchyma a peculiar brown or reddish-brown color.

Hoffmann has very exhaustively investigated the occurrence of the cardiac-lesion cells in consequence of stagnation of the blood in cardiac affections, and especially emphasizes the importance of this finding for the diagnosis. In two cases of hemoptysis in phthisical subjects and in 15 cases of pneumonia no cardiac-lesion cells could be found in spite of repeated examinations; in 14 cases of congestive conditions in consequence of affections of the heart they could be demonstrated more or less plentifully 13 times; they were always quite evident and could be demonstrated without difficulty. Why these cells which are produced in such large numbers in the lung of heart disease are not found in the frequent discharges of blood in pneumonia and phthisis, is explained by him as follows: In the former cases the blood gets to the surface at once, is subject to the influences of the air, the secretion of mucus, and the shocks of cough; and therefore has no opportunity to undergo the different stages of transformation. In heart disease, on the other hand, the red blood-corpuscles, partly by diapedesis, partly by capillary hemorrhages, enter the pulmonary tissue, are deposited under the protection of the epithelial cells, and thus there is a possibility of pigment-formation as in old hemorrhages within the tissues of the body.

Sommerbrodt does not believe in this explanation; he claims to have proved that alveolar epithelia contain such pigments, if blood is infused experimentally into the lungs through the trachea. Lenharz, on the other hand, considers the overwhelming majority of brown pigment cells to be more or less altered leucocytes containing blood-corpuscles. I, for my part, maintain here, as in regard to the entrance of coal pigments, the exclusive participation of the alveolar epithelium. The cells which are contained in the sputum and in anatomic specimens, and which are filled with brown pigment, are of so considerable a size that this alone precludes their identity with white blood-corpuscles. There is nothing to support the presumption that a gradual transformation of white blood-corpuscles into pigment-holding cells of such size can take place in the alveoli, much less the possibility of a place of formation outside of the alveoli.

If cardiac-lesion cells are mixed with the sputum in larger quantities, the latter assumes a very peculiar appearance. It can be best compared with frog spawn; only with the difference that the single dots in the gelatinous ground-mass are not black, but brownish-yellow.

Other objective signs are not necessarily associated with the presence of cardiac-lesion cells. It is true that the condition cannot be due exclusively to dilatation of the alveolar capillaries; some blood must escape into the alveoli, and in some places leucocytes also may be present, but the exudation is not sufficient to produce any notable change in the percutory signs. Bamberger, who made an accurate study of the clinical conditions in this process, states that in such cases the percussion note over the entire thorax is distinctly less resonant than normal, while the breath-sounds continue to be vesicular, but with a slight catarrhal element. In two cases, that of a little girl five and a half years old, and the above-mentioned case of a man nineteen years old, I myself observed complete dulness with catarrhal sounds over the left upper lobe.

DIAGNOSIS AND PROGNOSIS.

In the secondary form of chronic pneumonia, such as results from desquamative pneumonia associated with tuberculous infiltration of the pulmonary apices (see page 637), or follows other focal diseases, the diagnosis is possible only when the primary focus is small and the surrounding chronic inflammation extensive. Except under these conditions the diagnosis is of no particular importance. The subsequent course and the duration of the disease, however, cannot well be predicted without a knowledge of the diagnosis. For, although complete recovery may not take place, the expectation of life is greater in chronic pneumonia than it is when destructive processes are present in the lungs. It is from the latter, therefore, that chronic pneumonia must be chiefly distinguished. In this case it is primarily to be considered that in chronic pneumonia all sounds are absent in auscultation and percussion which are characteristic for material disintegrations of the pulmonary tissue, and only dulness of mostly moderate degree and catarrhal manifestations exist. It is also possible that tubercle bacilli are absent even with frequent examination of the sputa and in spite of the presence of tuberculous foci in the apices, if these are, so to say, encapsulated by the chronic pneumonia and, in proportion to the latter, only occupy a small part of the upper lobe.

Primary chronic pneumonia, if it sets in acutely with symptoms conforming to croupous pneumonia, as has been the case in the observations of Marchand and Fraenkel, cannot, until later in the course of the disease, be distinguished from other processes that sometimes accompany acute pneumonia. These are: pleural, interlobar, and mediastinal empyema, and abscess of the lungs.

The most frequent termination, empyema, may be considered first. It will not be necessary here to discuss the diagnostic signs nor the question of exploratory puncture. At the time when the onset of empyema is to be considered, there is generally no question of the inflammation having passed into the chronic stage, because the characteristic signs of this process do not appear plainly until during the further course of the disease.

After affections which originate in the pleura have been excluded the occurrence of abscess formations within the pneumonically affected lobe should be considered in the diagnosis. Only the examination of the sputa for elastic fibers can here be of value. They are sure to be found if only looked for.

I have to leave it undecided how much difference exists between pneumonia with delayed resolution—as described by Leyden—and chronic pneumonia, and whether there are sufficient diagnostic signs to make the distinction possible. At present all anatomic criteria are wanting for the discrimination of the two processes; even the case described by Leyden which ended fatally, and which he regards as the "anatomic prototype of delayed resolution," is undoubtedly a case of chronic pneumonia. The right lung was in a state of firm red hepatization with a granulated cut surface; the alveoli contained little air. Microscopically the alveoli appeared "mostly filled with coarse, fibrinous, net-like plugs," while in others the contents were less compact, and contained an abundance of pus-cells and alveolar cells, the latter sometimes in a state of fatty degeneration and of disintegration.

When chronic pneumonia is due to stagnation of blood in the lesser circulation, and the upper lobes are involved, it must be distinguished chiefly from tuberculous infiltration, which is more frequently associated with cardiac lesions than is commonly supposed. The diagnosis from acute pneumonic processes need not be considered, because the pulmonary changes that follow congestion in the course of the lesser circulation never develop acutely.

Regarding chronic consolidations, partly the presence of valvular disease, partly that of tubercle bacilli, will bring enlightenment, but no absolute decision, because a tuberculous infiltration may exist in cardiac valvular disease.

It follows from the above observations that the prognosis in all forms of chronic pneumonia must be regarded as unfavorable so far as complete restoration to health is concerned; but life may be prolonged for a considerable time, in spite of extensive contraction of the tissue, even if the entire half of a lung is involved. The acute disease, however, with an onset like genuine pneumonia, may terminate in death within a few weeks, especially if the process spreads from one lobe to the other.

TREATMENT.

We are able to combat the disease as such only with hygienic measures. If it manifests itself after the termination of the acute stage, or has developed gradually, the first requisite is air which is pure and free from dust, hence a sojourn in a wooded country is to be advised; the patient should avoid the influence of sudden changes of temperature, and give up every kind of physical labor. In regard to symptoms, it is to be remarked that the cough should be checked or at least relieved as much as possible, in order to prevent the disturbances of circulation connected with it. This is best done by means of morphin or Dover's powder. Against abundant expectoration of blood it is best to use lead acetate. In the presence of valvular defects digitalis is the best remedy to reduce too high a pulse frequency and all the consequences connected with it.

CARCINOMA OF THE LUNGS.

PATHOLOGIC ANATOMY.

Bayle had included carcinoma of the lungs among the six kinds of pulmonary phthisis which he described. Laennec, however, regarded it as a special disease, and designated it encephaloid on account of the consistency and the appearance of the lung. The macroscopic condition has been exemplarily described by him as follows: "The brain-like mass, at the height of its development, is homogeneous, milk-white, and resembles the medullary substance of the brain. In places it is rose-colored, and, in thin slices, transparent. The tissue is mostly less resistant than brain-substance. It is readily torn and broken with the finger. If the neoplasm is very large, one sees on its surface a moderately large number of blood-vessels the twigs of which penetrate the diseased tissue. As the vascular walls are very thin and delicate, they tear easily; this causes extravasations of blood, which may be found within the tumor-mass in the form of large clots. During the further course the carcinomatous mass undergoes softening."

The classification of pulmonary carcinoma into three kinds, as recommended by Laennec, does not require any further discussion. The classification proposed by Stokes has also been discarded. The disease, according to his statement, occurs in two forms: In the first as a degeneration of the lung; the latter being transformed into a carcinomatous mass without the formation of a tumor. In the second form the scirrhous or encephaloid mass forms a tumor which primarily is outside of the lungs, but finally causes displacement of the organs.

Rokitansky's classification is worthier of consideration. Cancer of the lungs may occur: (1) In the form of spherical, isolated masses, varying in size from that of a hemp-seed to that of a man's fist; these masses are inclosed in a delicate cellular envelope, while the substance of the tumor may resemble a mixture of jelly and bacon, a mixture of bacon and bone-marrow, or, finally, the medullary substance of the brain. The surrounding pulmonary tissue is displaced and, in common with the tumor itself, in a state of compression. This form is very rarely primary; other carcinomatous growths, usually a number of them, being present at the same time in several organs of the body. (2) As a special form of tubercle or nodule, about the size of a millet-seed or a hemp-seed, in association with cancer in some other organ. (3) In very rare instances the carcinomatous mass infiltrates the pulmonary cells and becomes diffuse; it is then the product of a pneumonic process which, chiefly under the influence of a dyscrasia induced by the extirpation of a cancer, manifests the external characteristics and elementary structure of carcinoma. There appears to be a hepatization of the lung with carcinomatous material.

This classification leads us immediately to the as yet unsolved question of the localization of carcinoma of the lungs, or to the question: In what histologic parts of the lungs does the carcinomatous affection originate?

In the first place, it is certain that carcinoma of the lungs may be secondary to carcinoma in distant organs; for instance, in the ovaries, pancreas, or liver. This is principally the case if some of the cancer nodes which are present in the above organs undergo softening. Then small particles of the tumor may get into the finer twigs of the pulmonary arteries by way of the veins or the lymph-vessels through the right heart, grow through the vascular wall with or without infarction in the sections supplied by these vessels, and form nodes of different sizes.

Like a great many authors, the names of which have been given by von Recklinghausen, I had the opportunity of observing a typical case of carcinoma of the lung from embolism of a pulmonary artery. This case deserves mention, particularly because the entire neoplasm did not extend beyond the adventitia of the vessel which contained the carcinomatous embolus. The latter consisted of a conglomeration of globularly arranged nests of concentrically stratified cancer cells; a few nests of cells in every respect identical penetrated the vascular wall as far as the adventitia. The width of the blood-vessel at the site of the embolus was about equal to the thickness of a heavy knitting-needle. The pulmonary section which corresponded to the distribution of the vessel was as large as half a plum and the seat of an infarct. The carcinomatous embolus had originated in a carcinoma of the gall-bladder which was as large as a fist and was mostly softened.

The second way in which carcinomatous degeneration of the pulmonary tissue may be brought about is by continuity. Then the primary seat of the carcinoma is the mucous membrane of the esophagus or that of the large bronchi. As growth continues, large nodes forming a uniform tumor-mass, which may reach the size of half a pulmonary lobe and more, then develop in the pulmonary tissue. Pässler has communicated quite a number of cases of this class in his very careful paper on primary carcinoma of the lungs which he based on 70 cases collected from the literature and 4 observations of his own. Ebstein also described one case very minutely, and Langhans, from the histologic examination of his case of primary cancer of the trachea and bronchi, came to the conclusion that the mucous glands were the starting-point of the entire carcinomatous formation.

In the third form of so-called primary carcinoma of the lungs the greatest difficulties and differences of opinion exist in relation to the determination of its origin.

Primary carcinoma of the lung is a diffuse carcinomatous affection of at least an entire pulmonary lobe, besides which nothing but a few diseased mediastinal glands of insignificant size are found. The few observations which have been made up to the present time do not permit of any doubt that such primary carcinomas actually occur, and that they originate, not in the bronchial mucous membrane, but in the pulmonary tissue.

After Rokitansky, who has described the manner of origin of this form very characteristically, Perl, by reason of his investigation of a case which, it is true, was not a strictly primary pulmonary carcinoma, according to his description, but started in the choroid, was the first to assert that primary carcinoma originates from the epithelium of the pulmonary alveoli. In this case, besides numerous nodules on the pleura and in the lungs, the anterior right lower lobe of the lungs was almost entirely transformed into an airless bluish-gray tissue, partly very flabby, partly permeated by firmer infiltrations. In the tissue, close together, were the thick-walled bronchi, filled with abundant tough, gelatinous, gray secretion. The microscopic examination revealed in the center of the infiltration masses of typical epithelial cells, partly concentrically stratified, and between them also broader connective-tissue trabeculæ formed by homogeneous fibers and at some places containing small round nuclei. "The supposition of a direct transformation of the pulmonary alveoli into carcinomatous alveoli; of the cells which in catarrh fill the alveoli into carcinomatous cells, has much in its favor."

Without very decidedly defending the alveolar origin of primary pulmonary carcinoma, Cornil and Ranvier have also maintained that in this affection the alveoli are filled with large, circular, or, through mutual pressure, polygonal cells, and that the alveolar walls mostly remain intact or are but slightly thickened by small round cells between the fibers. There is consequently no newly formed stroma in pulmonary carcinoma; the normal framework of the tissue stands in its place.

Malassez, after examining a case of primary carcinoma of the lung, positively declares the origin to be epithelial. At those places where the first changes were visible there was an epithelial neoplasm on the inner surface of the alveoli of the lungs. The cells were decidedly polymorphous. Sometimes they formed a layer of flat cells; at other times several layers of cylindric cells; now and then they were stalked and contained several large nuclei in the voluminous portion which projected into the alveoli after the manner of vegetations. Whenever the alveolus was completely filled with cells, it looked exactly like a carcinomatous alveolus. Furthermore, in the finest twigs of the bronchi and in the peribronchial connective tissue newly formed epithelial cells were also found which communicated with those of the alveoli. Continuations of the epithelial elements into the lymph-passages were also visible.

Grünwald gives a very detailed microscopic description of his case of primary squamous carcinoma. On the strength of it he comes to the conclusion that the malignant tumor originated in the alveolar epithelium because: (1) In sufficiently preserved alveoli a continuous connection between normal alveolar epithelium and cancer-cells could be demonstrated; (2) the cancer-cells throughout normal pulmonary epithelia; (3) the best preserved and therefore youngest cells were found on the alveolar wall, indicating that the latter is the starting-point; and (4) the connective tissue which contained the vestiges of the original normal condition, namely, particles of coal-dust, in an excellent state of preservation in spite of the beginning tumor formation, could not be the source of the proliferative process.

Boix has also described a case of primary carcinoma in which he regards the alveolar epithelium as the starting-point of the neoplasm. He says distinctly that he does not care to decide the question whether the cancer stroma is formed exclusively from the pulmonary tissue itself. For, although the framework of the peripheral sections was delicate and plain enough to indicate that the pulmonary alveoli were normal, in the center of the tumor there existed without doubt a plentiful new-formation of connective-tissue trabeculæ under which the epithelial cells had almost disappeared.

These communications should suffice to prove that the alveolar space, and a transformation, if not a substitution of the alveolar epithelium, may be regarded as the origin of primary cancer. It does not appear to me as indicated, in consideration of the object which I had in view with this work, to enter upon further investigations in regard to the origin of primary carcinoma of the lungs, especially the participation of the lymph-vessels (Schottelius). I content myself with citing the résumé given by Siegert, which is based on the ample literary and his own material and reads as follows:

Primary epithelial cancer may develop: (1) From the alveolar epithelium; (2) from the epithelium of the bronchial mucous membrane; (3) from the epithelium of the bronchial mucous glands.

Primary endothelial cancer may develop: (1) From the endothelium of the superficial pleural lymph-vessels; (2) from the endothelium of the deep pulmonary lymph-vessels.

Specially to be mentioned is the occurrence of carcinoma in tuberculous portions of the pulmonary tissue. Friedländer found a cancer of more than hazelnut size on the wall of a tuberculous cavity in the lower part of the left upper lobe of a tuberculous lung continuations of which protruded into the left main bronchus. He refers the origin to a new-formation of stratified pavement epithelium which develops on ulcerated areas.

Hildebrand, Siegert, and Ribbert made similar observations. The latter concluded that there is a special disposition of the cicatricial tissue to epithelial proliferation from the fact that the tumor develops in chronically inflamed pulmonary tissue.

Bayle has already demonstrated the association of tuberculosis and carcinoma in the lungs. Among 31 cases of carcinoma, described by Wolff, 23 were complicated by tuberculosis. Among 10 cases of primary cancer of the lungs, on which he performed the autopsy in the Friedrichshain Hospital, Schwalbe was able to demonstrate the coexistence of tuberculosis in the same lung in 3 cases. Tubercle bacilli were found in each case.

After stating the histologic origin of pulmonary carcinoma, it remains to say, regarding the gross pathology, that the disease generally spreads from the hilum of the lungs or attacks the greater part of one pulmonary lobe, or even an entire lobe, more rarely all the lobes of one side. In such cases the other lung almost always remains free. If the upper lobe is attacked, the disease seems to spread last to the apex, so that, at the autopsy, the latter may sometimes be found free from carcinomatous masses and containing air (Reinhard).

Owing to the gradual spread of the neoplasm from the hilum of the lung, the disease, as Reinhard has pointed out after a thorough statistical investigation, advances in most cases along the bronchial branches, rarely along the pulmonary blood-vessels. In the former case, smaller branches of the bronchi frequently become constricted; even fairly large bronchi may terminate blindly in the tumor-mass.

If the disease is diffuse and involves a large part of an entire lobe, the central portion often undergoes softening, the process being favored by the presence of secretions and pathogenic materials in bronchi that have become impervious. This may be followed by extravasations of blood and hemorrhages.

Almost always the mediastinal glands will be found to be considerably swollen. They are of essential importance for the formation of the clinical picture. Rarely one or several of the supraclavicular glands are affected, generally only, if the seat of the carcinoma is in the upper lobe. Then they are conspicuous by their remarkable size. Still more rarely the lymph-glands of the axillary space will also be affected.

The pleura is sometimes affected by purely inflammatory processes which lead to effusions mostly of a hemorrhagic or purulent nature.

ETIOLOGY.

Whether the remote cause of pulmonary carcinoma, as well as that of carcinoma in general, depends upon local conditions (antagonism of the tissues, pre-existing nuclei), or whether it is to be attributed to invasion of the tissue by parasitic formations, an important immediate cause of the pulmonary carcinoma I take to be the effect of severe trauma, which, however, does not produce laceration of the pulmonary tissue, but only molecular disturbances of an unknown character. At least I cannot regard it as merely accidental that in the four cases of primary pulmonary carcinoma, which I had the opportunity of seeing, grave trauma had preceded. In one case the woman had fallen from a ladder on the right side of her breast while cleaning windows, and, since then, had continually complained of pains in the right side of her breast. Sixteen months later she died, thirty-six years old, of carcinoma of the entire right lung, as was demonstrated at the autopsy. The second case was that of a man, forty-six years of age, who, with others, was lifting a heavy beam, when, through a misunderstanding, the others let go, and the full weight of the beam rested on his shoulder alone. Two years later he died of diffuse carcinoma of the left upper lobe.

Georgi has also described a case of medullary carcinoma of the left lung, the origin of which he traces to trauma. A piece of iron weighing about eight pounds broke when hammered and was thrown against the front and left side of the chest of a blacksmith sixty years of age. He died a year later. Georgi explains the fact that the formation of tumor took place above the spot where the man had been hit —*i. e.*, in the upper lobe—by assuming that just at the affected spot the lung was compressed, and the air and blood were driven out; while above this place, a strong congestion or slight lacerations of vessels took place, so that there a lasting injury was inflicted and the strongest inflammatory reaction caused, which in its further course offered favorable conditions for the development of the neoplasm.

Löwenthal demonstrated a primary carcinoma in the left lung of a man thirty-two years of age about eight months after a "kick" against his chest.

It is also worth mentioning that pulmonary carcinoma occurs more frequently in men than in women. Out of 22 cases collected by Hasse, only 5 were women, 17 men; of Reinhard's 27 cases, 11 were women, 16 men.

Remarkable is also the occurrence at a comparatively early age. Of the above 22 cases of Hasse, 9 occurred between twenty and twenty-nine years of age, 8 between thirty and thirty-nine, 2 between forty and forty-nine, 2 between fifty and fifty-nine, and 1 between seventy and seventy-nine. "Consequently the greatest disposition is shown during the prime of life."

Reinhard observes, opposed to these statements, that, according to his compilations from the books of the Dresden Hospital, out of 70 cases only 15 were under forty years of age, while 55 were over. But it seems that his figures are not quite reliable, as a later compilation by Fuchs from the records of autopsies held at Munich, according to the statement of Grünwald, shows that these tumors differ materially from cancers in other parts of the body. Out of 60 cases, 11 were between twenty-one and thirty; 10 between thirty-one and forty; 12 between forty-one and fifty; "therefore over one-third of all cases occurred at an age which is generally considered immune from carcinoma."

SYMPTOMS.

The clinical manifestations of pulmonary carcinoma are extremely dissimilar, in keeping with the variety of the described pathologic processes. To be able to give an approximately accurate picture I consider it necessary to distinguish—

1. Primary carcinoma, originating in the pulmonary tissue,—or, more correctly, in the alveoli,—occupying a more or less extended part of a pulmonary lobe, or an entire lobe, or even an entire lung.
2. Carcinoma originating in a larger bronchus, at the onset mostly occupying those portions of the pulmonary substance which are in the neighborhood of the hilum and spreading from there to the periphery.
3. Secondary carcinoma, originating in remote organs, or, more correctly, isolated carcinomatous nodes of different sizes appearing in the lungs.

The first-named form is principally characterized by a number of physical manifestations which I cannot describe better than by quoting Reinhard:

"Among the primary objective symptoms that are observed on examining the patient the demonstrable alterations in the thorax and in the lung first arrest the attention. After the disease has lasted some little time, the thorax on inspection presents a noticeable asymmetry of its two halves, as regards either its dimensions or the degree of curvature. Sometimes a change of the three dimensions, and with this of the thoracic circumference of the affected side, is most prominent; then, again, the dimensions on both sides are equal, but in viewing the anterior wall of the thorax it is noticeable that the two halves show differences of curvature. This depends more or less upon the parts of the lungs in which the carcinomatous growth predominates. If the principal seat of the disease is located in the lower parts of either lung,—*i. e.*, in the lower and middle lobe,—there will be, as in pneumonia and pleural exudations, deviations in the circumference of these parts; if, however, an upper lobe is the principal seat of the new-growth, an alteration of the thoracic arch will be noticed in this part. In what way this deviation from the normal takes place depends upon the manner of extension and the proportions of growth of the tumor, and upon other anatomic conditions. If proliferation is very conspicuous, an increase manifesting itself in considerable extension and in obliteration of the intercostal spaces of the affected side is the necessary consequence. If, on the other hand, the interstitial inflammation, which progresses *pari passu* with the growth of the neoplasm, and is accompanied by shrinking of the tissue and the formation of hard callosities that undergo cicatricial contraction, predominates, these structural changes find their clinical expression in a negative modification of the normal curve, or, in other words, in retraction of the thoracic wall. Accordingly, the statement will be found in some cases that the affected side of the thorax is widened or narrowed or finally retracted; but any of these deviations may be wanting. Exact information about these conditions is gained by repeated measurements and comparisons of the diseased with the healthy side of the thorax. A decreased expansibility of the affected pulmonary parts is determined partly by inspection, partly by application of the hand during deep inspiration."

If in primary carcinoma only the lower lobe is affected, every characteristic symptom may be absent, according to my experience, and the impression may be created at the first moment that one has to deal with a simple pleural exudation. The objective symptoms are remarkably like those of pleuritis. Exploratory puncture will, of course, be negative, and, upon close examination, the dulness on the anterior thorax reaches higher than in an uncomplicated pleuritic exudation. The following observation, in which the anamnesis did not give any support for the recognition of the disease, will best tend to prove this:

Mill-owner B., sixty-five years of age, two years before his last disease was suffering from a disease of the bladder, which was cured in a short while. At the end of October, 1885, after hunting, he was attacked by asthmatic symptoms. Two weeks later there was dulness in the right side, corresponding approximately to the middle and lower lobe; at the same place weakened respiration; no pectoral fremitus. The percussion note over the upper lobe was very variable,—*i. e.*, sometimes more, at other times less loud,—the respiration weak but purely vesicular. The patient was pale, but well nourished. The temperature did not rise above 38.2° C. (100.8° F.) in the evening, the pulse was very frequent. Asthma did not, however, appear during the course of the disease when the patient was in bed. The sputa were not very abundant, dark, tough, mucopurulent, and rusty. Although the dulness on the anterior thorax reached higher and was more intense than on the posterior side, the possibility of an empyema was thought of; but aspiration with the Pravaz syringe only yielded blood. Two weeks after the onset of the disease, with gradual decline of strength, death occurred. The autopsy showed a diffuse carcinoma (medullary sarcoma) of the lower lobe and a part of the middle lobe, carcinomatously degenerated mediastinal glands, and a stone in the bladder the size of a pigeon-egg.

The possibility of mistaking this condition for pleuritis is comprehensible if a patient comes under treatment under circumstances or at a time as described in the following observation:

Agent W. H., fifty-eight years of age, has suffered since early in 1886 from cough and asthma. As he thought that his first physician had not examined him sufficiently after the disease had existed for a year and three months, he went to another one who caused him to go to a hospital at once, because an immediate evacuation of his left-sided pleural effusion was necessary.

The patient is weak in structure, slightly pale, weight 55 kg. His father died of cancer of the stomach, at the age of sixty-five. Except for the disease which brought him to the hospital, he has always been healthy. The examination shows a dulness over the left posterior side of the thorax from the middle of the scapula downward; over this part respiratory sounds and pectoral fremitus are not present. In the line of the left axilla the dulness reaches almost as high as posteriorly. Only anteriorly above the heart loud, full sounds are present. The case was taken to be one of simple serous effusion and salicylic acid was prescribed. As this proved to be without any effect, the dulness even advanced upward, and the asthma increased, an aspiration was performed three days later, on March 18th, and 2300 c.c. of a serous fluid with a slight tinge of blood was discharged. After this the dyspnea decreased, but the dulness continued over the left lower lobe. During the next days it advanced still higher and the dyspnea increased again. On March 24th, suddenly edema of the lungs set in with fatal termination.

The autopsy showed: Medium-sized, poorly developed body with slightly pale yellow skin, livid lips, weak, pale muscular structure, and very scanty cushions of fat. The pericardium contains about two tablespoonfuls of bloody-serous fluid. The heart is of normal size, the muscular structure is brownish-yellow, pale, flabby; all valves are intact. The left pleural cavity contains a liter of bloody-serous fluid. The lower lobe of the left lung consists of a completely airless, dense white mass in which only the largest bronchi can be distinguished. The middle lobe is softened and transformed into a greasy, yellow, almost purulent looking mass. The upper lobe is small and contains but very little air. The right pleural cavity is empty, the right lung very edematous and hyperemic, otherwise without change.

The spleen is of normal size, firm on section, of brownish-red appearance.

Both kidneys are very hyperemic, otherwise normal. The liver is of normal size. On the surface as well as on the cut surface a great number of firm, whitish nodes ranging in size from a millet-seed to a hazelnut are present. On the mucous membrane of the small intestine swollen solitary follicles are visible.

As can be said afterward, in this case the hereditary disposition, the area of dulness over the left lung, reaching almost as high in the axillary line as posteriorly, the slightly bloody appearance of the aspirated fluid, and especially if, in view of these suspicious signs, the posterior thorax had been examined immediately after the evacuation of 2300 c.c., the persistence of the dulness might have suggested the diagnosis of carcinoma.

In the second class of cancers—*i. e.*, those which originate in the bronchi—a greater number of symptoms are observed, principally because these growths produce narrowing of the air-passages, and may be accompanied by glandular swellings, especially enlargement of the mediastinal glands which often attain a considerable size.

Either stenosis of the air-passages is caused by carcinomatous proliferation within the substance of the mucous membrane, or the trachea and bronchi may be surrounded by carcinomatous masses, so that the tubes are compressed from without. Clinically this leads to whistling, long-drawn-out respiration, both phases being equally labored. In the case of a patient who had been admitted for a large, right-sided empyema which was evacuated by operative means, I once had the opportunity of making the diagnosis of pulmonary cancer, and my diagnosis was largely based on this very inspiratory and expiratory stridor.

The case was that of a man forty-six years of age, who said that during the eight months preceding his admittance to the hospital on May 7, 1892, he had had pleuropneumonia, and had suffered from great difficulty in breathing for the last four months. On the day named three liters of thick pus, mixed with abundant large fibrin coagula, were evacuated from the right pleural cavity by resection of ribs. Only when after this operation high-graded stridor and inspiratory and expiratory dyspnea remained, the diagnosis of carcinomatous affection of the right lung could be made on account of the other symptoms present—namely, enlarged supraclavicular glands, the finding of a tumor as large as an apple in the lower section of the left lobe of the liver, occasional rusty sputa, and slight paresis of the right vocal cord. Fever had never been present. Twenty-six days after the operation death followed. At the autopsy a more than fist-sized medullary carcinoma was found in the mediastinum which was near the pericardium; the right lung, which had been compressed, was collapsed, airless, and contained numerous carcinomatous nodes. The mucous membrane of the lowest part of the trachea and of the right bronchus contained flat nodes, and the lumen of these ducts was much stenosed. The tumor in the left lobe of the liver, which had been observed during life, likewise proved to be a medullary carcinoma.

From the same cause, stenosis of the trachea and both main bronchi, is to be explained the decrease or cessation of the very loud, harsh, bronchial respiration which under normal circumstances is audible over the trachea immediately above the manubrium during the entire duration of inspiration and expiration. Of 4 cases, in which I had the opportunity to observe this phenomenon,[14] 2 were mediastinal carcinomata which had grown into the trachea exactly at the bifurcation and gradually closed the entrance into both bronchi.

I explained the change of the normal sound over the trachea as follows: The production, under normal circumstances, of bronchial respiration by the air passing from the narrow glottis into the wide trachea is due to the formation of eddies beyond the stenosed place —*i. e.*, beyond the glottis. But this requires a certain velocity of the air-current, which under normal circumstances is secured by the almost uniform width of the entire trachea. If the latter, however, is narrowed in one place, so that its lumen is only as wide as the glottis or less, the eddies of air must become less in the passage from the glottis into the trachea, and consequently the normal bronchial respiration undergoes the described modification which is valuable for the diagnosis of tracheal stenosis.

Of course, it still remains to be demonstrated in what cases and in what kinds of stenosis of the large air-passages this change of the bronchial respiration takes place. In a case of Jacoby of aortic aneurysm with syphilitic tracheo-bronchostenosis, of which all the symptoms were present, and which was also indicated by the laryngoscope plainly showing visible elevated curvature of the tracheal wall, the respiratory sounds over the trachea were perfectly normal. He considers it not impossible that the respiratory sounds over the trachea of his patient continued, because there existed not a constriction of one place in the trachea, but a duct-like stricture continuing into the bronchus.

Furthermore, symptoms due to pressure on the nerves and vessels in the mediastinum are to be considered. Pressure on the recurrent laryngeal nerve must lead to paresis of the vocal cord, in the esophagus, and to difficulties in deglutition; an obstruction of the venous reflux to the heart may be followed either by bloating of the supraclavicular parts or dilatation of the subcutaneous veins over the upper parts of the thorax, or edema of one arm.

In the two cases of primary carcinoma of the lungs described by Kasem Beck an intense enlargement of the cutaneous veins had set in only on the side corresponding to the diseased lung. If glandular enlargements also take place in the supraclavicular parts, they never reach any considerable size.

Besides all these symptoms, a more or less extensive area of dulness over the lungs, mostly over the upper lobes, must be present. The dulness may alternate or be associated with a great variety of percutory phenomena. Thus, Woillez mentions "*tympanisme thoracique*" as a not exactly characteristic but important symptom of pulmonary cancer. It is a tympanitic sound which, when a tumor is present in the pulmonary tissue, sometimes precedes the dulness depending upon it, and which is produced by the pulmonary parenchyma over and next to the tumor being still pervious to air, but relaxed and not able to assume its usual tension. Grünwald describes a very remarkable change of the physical signs in his case. Sometimes the dulness was replaced by a tympanitic note with bronchial respiratory sounds, at other times this change occurred only in the dorsal position, sometimes the extent of the dulness decreased, at other times it increased. Ebstein lays stress upon the importance of conspicuously regular, rapidly changing zones of dulness.

The complete obstruction atelectasis of the right lung, as described by Körner, deserves to be mentioned as a rare consequence of a bronchial carcinoma.

The least significant symptoms are present in carcinoma secondary to carcinoma of remote organs, because they rarely reach considerable size. The carcinomatous cells are, as mentioned, undoubtedly transferred by means of the vascular system. Mostly they get into the lesser circulation, and from here like emboli into the branches of the pulmonary artery.

The character of the sputum is the only symptom that is practically the same in all the various modifications of the process, depending on origin, seat, and size of the tumor. The greater the extent of the carcinoma, the more readily will the peculiarities of the sputum be observed. In addition to the occasional admixture of blood, which in itself presents nothing characteristic, even when it resembles raspberry or currant jelly, the sputa of these patients contain certain cellular structures which, according to Hampeln, who was the first to mention them, are a sure sign of the presence of cancer.

These structures are unpigmented, polymorphous, polygonal cells, varying greatly in size, with distinctly outlined nuclei and equally distinct nucleoli. They may be agminated or solitary, and are sometimes of gigantic size. He believes himself justified in concluding that "these structures, which do not occur under any other circumstances, are produced by a neoplasm," on the ground that the normal polygonal epithelial cells of the pulmonary alveoli, "unpigmented" and unchanged as to shape, if they occur in the sputum at all, are found only in isolated examples. The presence of these cells, however, tells us nothing in regard to the seat of the cancer; above all, they do not negative the existence of a bronchial carcinoma.

Betschart reports a similar finding in a case of pulmonary carcinoma. In fresh sputa large, circular cells were found, lying close together, with one to four nuclei, each of which contained one to four nucleoli. The shape of these cells was round, oblong or polygonal with rounded corners; some were of remarkable size. Their protoplasm was finely granular; the granulation in the surrounding of the nucleus was slightly denser than in the peripheral zone, which made the latter appear paler.

Ehrich reports the finding of tumor particles in the sputum. A man fifty-one years of age, about a year after the onset of the disease, expectorated, besides blood, villous masses which, according to Marchand's investigation, consisted chiefly of numerous polymorphous cells, which were often arranged in large clumps, but sometimes showed a kind of concentric stratification. At one place a group of polyhedral cellular forms was found with very large bloated nuclei.

Krönig succeeded, by puncture with a cannula the diameter of which was a little larger than that of an ordinary Pravaz syringe, in drawing out a piece of yellowish-white substance, the microscopic examination of which showed a conglomeration of lymph-corpuscles with very large nuclei. They were embedded in a very fine reticulum, and at some places surrounded by bundles of elastic fibers from which the presence of a malignant new-formation could be concluded. The autopsy showed a carcinomatous sarcoma.

Of other characteristic symptoms of pulmonary carcinoma, and carcinoma in general, remain to be mentioned marasmus and intermittent fever. Both manifestations have been exhaustively discussed by Hampeln.

In relation to the former, he remarks that every visceral carcinoma manifests itself either in the form of marasmus which appears when poverty, want, misery, and disease complicate old age, or by the signs of profound anemia. But these types are not possessed of anything specific.

It is surely correct that anemia may be caused by carcinoma, but according to my experience, it can be of service for the recognition of carcinoma at most in the very last stages.

Regarding the intermittent fever, so far as it is to be made of importance for the recognition of pulmonary carcinoma, Ebstein remarks correctly that it is much more likely to indicate tuberculosis, even if no tubercle bacilli are present in the sputum; for the temperature not very rarely presents this type in this comparatively more frequent affection.

DIAGNOSIS.

In the diagnosis, as in the symptomatology, the problem must be viewed from a different standpoint according as we are dealing with diffuse, parenchymatous carcinoma of the lower lobe or of an entire lung, or with a new-growth originating in the upper lobe or in the bronchi. In regard to the former, the error of mistaking it for a simple pleurisy must be especially guarded against. The mistake is quite pardonable and may easily be made in the early stages of the disease, before any marked symptoms of cachexia have developed. The history may fail to give any clue whatever, as in the case described above, on page 715, in which the patient attributed his illness to a hunting expedition, during which he thought he had caught cold.

When making a physical examination, it is to be borne in mind that a dulness over the lower lobe due to carcinoma extends higher than dulness due to a pleural effusion. The question must be decided by an exploratory puncture which gives a negative result, or, if a large needle is used, sometimes brings forth particles of tumor. The diagnosis becomes still more difficult if a pleural effusion sufficient to explain the existing symptoms is present. At the same time the fact that the fluid is mostly not purely serous, but mixed with blood, as in the case mentioned on page 716, is significant. A considerable admixture of blood nearly always indicates the presence of a carcinomatous or tuberculous process in the pleura or in the lungs, and accordingly all further diagnostic points have to be considered. If in spite of proper medication no resorption takes place, and a second thoracocentesis is indispensable, it will be necessary to determine, immediately after evacuation of the fluid, whether the dulness has entirely disappeared, which in strong patients may be done in the sitting, in weak patients in the lateral position; a persistence of the dulness after abundant discharge offers the best explanation of a doubtful case.

The diagnosis of diffuse sarcoma of the pulmonary lobe I may discuss in connection with the following case, which took a rare course:

The laborer, G. St., was admitted to the hospital on February 12, 1896. In June, 1894, his right thigh had been amputated on account of a bone tumor, about the nature of which nothing further could be learned. Up to ten weeks ago he was perfectly healthy. Then hemoptysis set in, which since then recurred frequently. During this time he also suffered much from night-sweats.

Condition on February 13, 1896: Patient is small of stature, pale, but in moderately good condition. He has slight fever; the urine contains no albumin. The upper third of the right thigh ends in an amputation stump. The abdomen is soft, liver and spleen are of normal size. The heart is not enlarged, the heart sounds are clear. The percussion sound over the anterior lung is full and deep, the respiration vesicular. Right posterior, from the spine of the scapula down, there is dulness, no respiratory sounds are audible; the pectoral fremitus has disappeared. Exploratory puncture brings blood-colored pus.

February 15, 1896: After costotomy ½ liter of pus mixed with blood is discharged from the right thoracic cavity. In the evening the patient is much relieved. The sputum is sanguinolent, contains no tubercle bacilli.

February 17, 1896: The secretion during the daily change of dressing is insignificant. Right interscapular dulness on percussion, and loud bronchial respiration. Over both pulmonary apices the sound is full and deep, the respiration vesicular. The previously severe cough has abated.

February 27, 1896: Yesterday there was more irritation, causing cough. The temperature rose to 39.2° C. (102.6° F.), the pulse was 132. To-day toward morning he perspired very much, so that he had to change his linen.

March 30, 1896: With very little fever, insignificant secretion from the puncture wound, occasional blood streaks in the sputum, frequent severe perspiration in the morning, a piece about 2 cm. length was resected from the vertebral end of the rib which touches the wound, on account of the carious condition of its inner surface.

April 24, 1896: The secretion from the puncture wound has nevertheless continued, even increased during the last days. It was discovered that the caries of the affected rib extends further to its central part; therefore, under chloroform narcosis, a piece 10 cm. long was resected. At the bottom of the wound decayed pulmonary tissue is visible. The wound was packed with iodoform gauze.

May 5, 1896: The wound is narrowing; there is no secretion. Right posterior, from the spina scapula down, dulness and absence of respiratory sounds; sometimes dry, barking cough; often mucous, slightly sanguinolent expectoration.

June 1, 1896: The wound has closed entirely. The subjective condition is good, but the dulness from the spine of the scapula down, the absence of respiratory sounds, and the diminution of the pectoral fremitus continue.

June 19, 1896: The sputa have not been bloody during the last days; the wound has completely cicatrized. The condition is the same as on June 1st. At his request the patient is discharged as cured.

On June 28th he has to be readmitted because dyspnea, insomnia, barking cough, rusty sputum, and diarrhea have set in. The percussion sound over the right thoracic side is dull anteriorly as well as posteriorly; respiration and pectoral fremitus are absent. The sputum shows admixture of blood; the cough is short, barking. The heart is normal. The abdominal walls are slightly tense; the liver extends about 5 cm. below the costal margin; the spleen is normal. No fever exists; the urine is free from albumin.

July 2, 1896: The patient has whistling inspiration. The liver constantly extends 5 cm. below the costal margin. Pressure over the liver does not produce pain. On the right side over the fossa supraspinata the percussion sound is empty and high in pitch; no respiratory sounds are audible. Dulness is present down to the base of the lung. The respiratory sounds here are also suspended; only in the interscapular space, on the right, very low respiration can be heard. On the right side in front and over the side of the chest dulness everywhere; the respiratory murmur is inaudible. The impulse of the heart can be seen and felt to the left, between the fifth and seventh intercostal spaces to the middle axillary line. Above, the heart dulness reaches as far as the upper border of the fourth rib. The right forearm and the right hand are edematous; the circumference of the former at the widest place is 25 cm., that of the left forearm 21.5 cm. The sputa are grayish-black, mixed with blood.

July 4, 1896: The edema in arm and hand has increased, the face is also edematous. Low, remote bronchial respiration is heard over the right anterior thorax. In the evening, with high-graded dyspnea and gradual diminution of the pulse, death occurs at 9 o'clock.

The autopsy was performed on July 6th. The left lower extremity, the right arm, and the face are edematous. The right thorax is in all dimensions wider than the left. After opening the abdominal cavity one sees the liver extending about 6 cm. below the costal curvature. After removal of the sternum and the costal cartilages the right lung practically presses forward. The heart is displaced far to the left.

The pericardium contains a small quantity of cloudy serous fluid. The heart is moderately large, the muscular structure coarse, pale, of normal thickness; in the cavities of the heart are ample cruor masses and lardaceous coagulations. The valves are all intact. The weight of the entire heart is 312 gm.

The right lung is adherent to the thoracic wall. Its weight is 2400 gm. The tissue is completely airless. Only in the apex of the upper lobe the structure can be recognized; otherwise upper and middle lobes consist of a uniform, whitish mass which in some places presents yellow and yellowish-red spots, at other places contains softened areas. The lower lobe on the cut surface has a uniformly yellowish appearance, which is interrupted by some gray hyaline markings. In the posterior lower part the appearance is more uniformly hemorrhagic. The principal vein of the lower lobe and a great number of its twigs are thrombotic. The left lung is nowhere adherent to the thoracic wall, contains much air, and is but little edematous.

The spleen is 15 cm. long, 8 cm. broad, 4 cm. thick, its tissue firm, pale brown on the cut surface; its weight is 190 gm.

The left kidney is difficult to remove from its capsule; 11 cm. long, 4 cm. broad, 3.2 cm. thick; its consistency is firm; the cortex shows normal conditions on the cut surface. The right kidney—12 cm. long 5.5 cm. broad, 3 cm. thick—does not show any abnormalities. The two organs together weigh 320 gm. The liver is much enlarged and weighs 2035 gm. The cut surface shows the typical picture of nutmeg-liver.

The mucous membrane of the stomach is swollen; the mucous membrane of the intestine is pale.

The microscopic examination of the diseased lung showed that the main mass of the tumor consisted of closely packed layers of round cells. In many places they were intact; *i. e.*, in applying the three-color stain the blue-colored nuclei showed distinctly in the brown protoplasm. In some places the nuclei were surrounded by a pale, blue circle, while the central larger part showed darker tints. But, besides, other parts were found where no nuclei could be distinguished in the cells; the latter, then, represented nothing but brown, circular, clearly defined structureless formations. At such places frequently many wide blood-vessels with delicate walls were present. The entire mass was interspersed with long and broad tracts of connective tissue, containing also elastic fibers, and intersected by oblong ducts of varying width which, according to the direction of the section, proved to be empty circular spaces. Their walls were thin and structureless.

Finally, it remains to be mentioned that in many places circular formations were visible which after staining assumed a golden appearance. These formations often showed fine wavy processes. (Compare Plate 6, Fig. 12.)

The above case demonstrates the remarkable clinical congruity of diffuse pulmonary sarcoma, if it originates in the lower lobe, with diffuse pneumono-carcinoma. The essential differences are found in the youthful age and in the existence of a primary neoplasm in a peripheral part of the body, in this case in the thigh. Whether in the future a careful examination of the sputa will render it possible to demonstrate the differences must be left undecided for the present. In either case the tumor may be hemorrhagic.

As is proved by this case, the complications are essentially the same whether the diffuse affection of the lungs be sarcomatous or carcinomatous. Hemorrhagic or purulent pleuritis is the most important complication to be considered. In the case in question purulent pleuritis was complicated by caries of the ribs which could only be removed by the resection of a piece of rib 10 cm. long. That against expectation the wound closed completely over the sarcomatously degenerated lungs, with the ragged appearance of that part which was visible through the resection wound, I am inclined to ascribe to a great extent to the influence of the iodoform gauze. I do not venture to explain the finding of the circular formations with fine, wavy processes illustrated on Plate 6, figure 12, although the curved shape of the spurs appears to indicate an organic growth which may be in an etiologic relation to the neoplasm.

Carcinoma of the upper lobe, and carcinoma originating in the bronchial mucous membrane—which, as has been mentioned, are almost without exception accompanied by a variable degree of swelling of the mediastinal glands—must be differentiated, on the one hand, from tuberculous processes, and, on the other hand, from tumors or aneurysms in the mediastinum. Regarding the former point, Hérard and Cornil say: "Cancer of the lungs, the general seat of which is the base or the middle of lungs, sometimes occupies the apex, and, if it has not softened, may then be mistaken for commencing tuberculous infiltration. But in cancer of the lungs the dulness is more considerable, the dyspnea and the subclavicular pains are more intense; at the same time there are symptoms of central compression, as is shown by the gradual suspension of respiration on the affected side; the expectoration may be absent or insignificant; if it is more abundant, it does not present anything characteristic, or it consists of pure blood or of gelatinous masses similar to currant jelly. Occasionally a supraclavicular gland may be demonstrated."

These diagnostic remarks I can supplement from my own experience. The presence of large hard glands in the fossa supraclavicularis in diagnostic relation indicates a carcinomatous affection. In tuberculosis of the upper lobes the glands are relatively quite small; if they are enlarged at all, they are soft and tend to break down.

Extensive dilatation of the veins over the upper anterior portions of the thorax are also in favor of carcinoma and against tuberculosis, but not against other tumors of the mediastinum.

With regard to the differential diagnosis from aneurysm and tumors which have developed in the mediastinum exclusively, it is to be remembered that all the before-mentioned symptoms, as far as they depend upon compression of the structures in the mediastinum (vessels, nerves, esophagus), may exist in these conditions, and that in aneurysm vascular murmurs (bruit), pulsations in abnormal places, delay of one radial pulse, may also be present. Absence of dulness over the lungs, especially over the upper lobe, and the absence of all changes in the sputa characteristic of carcinoma of the lungs, are the only available diagnostic points under these circumstances.

[Malignant growths in the lungs may be diagnosed by the sputum. Betschardt found large round cells arranged in bands, with one or more nuclei, in a suspected case, and upon these findings made a correct diagnosis.

The following case presented difficulties that are exceptional. The patient, a male aged forty-nine, after unusually hard work during the winter, to which he attributed some fatigue, was seized with an acute pleuro-pulmonary attack. Three weeks afterward there were physical signs of localized effusion at the right base, subcutaneous edema over the lower zones of the right thorax, moderate fever (101° F. in the evening), a leucocytosis of 14,000, and exhausting sweats. An exploratory operation disclosed a hard mass in the center of the lower lobe with infiltration of the remainder, which was taken to be a fibroid mass. The leucocytosis and fever continued. The liver was enlarged and the seat of a large smooth nonfluctuating mass. This proved to be a cancer of the liver, and the autopsy later disclosed the primary growth in the right lung. There had not been any hemorrhage.—Ed.]

TREATMENT.

As all endeavors looking toward the cure of cancer have so far proved unsuccessful, there is nothing to be said about the treatment of pulmonary carcinoma. Nevertheless, there is scarcely any other disease in which the physician's efforts are more indispensable. Not only the symptoms, such as dyspnea, cough, hemoptysis, but also complications, especially pleurisy and empyema, imperatively demand his speedy intervention. The physician may here have an opportunity to prove both his knowledge and his humanity.

EMBOLISM, THROMBOSIS, AND INFARCT.

PATHOGENESIS AND PATHOLOGIC ANATOMY.

I believe that I shall introduce the discussion of the above pathologic conditions best by asking the following questions:

Can infarct of the lungs exist without embolism and without thrombosis?

Is it possible for an infarct of the lungs to be the direct and immediate consequence of embolism?

Is it possible for an infarct of the lungs to be produced by local thrombosis in a previously normal lung?

Laennec has given an exemplary description of what an infarct of the lung really is. Its seat, its size, its appearance, have been correctly described by him. According to him, the condition manifests itself as a consolidation which is always partial and rarely occupies a large portion of the lungs; it generally occupies one to four cubic inches. It is always distinctly circumscribed; the engorgement is just as considerable where the consolidation ends as in the center. The surrounding pulmonary tissue is generally sound and crepitant, and does not show any resemblance to the consolidation in pneumonia, which gradually decreases toward the periphery. The tissue surrounding these hemoptoic engorgements is even frequently very pale, rarely pink, sometimes also red and infiltrated, or only colored by a small quantity of very red blood; but even in such cases there is a well-defined rectilinear demarcation between this hemorrhagic infiltration and the dense engorgement. Laennec attributed the process to a hemoptoic occlusion (*engorgement hæmoptoique*). The veins of the diseased parts he sometimes found filled with coagulated, half-dried blood, "a kind of infarct." He applied the term "pulmonary apoplexy" to the disease as a whole.

Rokitansky has confirmed Laennec's observations. He emphasizes especially that the hemorrhagic infarct need not be followed by hemoptysis. He says, further, that the apoplexy often coincides with a state of active dilatation of the right heart. He also exhaustively describes how the disease may terminate in recovery, the infarct shrinking to a cellulo-fibrous white or black tissue.

This pulmonary alteration was placed in an entirely new light by the theory of pulmonary embolism which was originated by Virchow. In regard to the relation between the condition of the blood-vessel and that of the pulmonary parenchyma, he points out the necessity of distinguishing between primary and secondary clot-formation in the pulmonary artery. In one case occlusion of the vessel precedes the change in the parenchyma and is independent of it; while in the other the occlusion is the cause of the parenchymatous alteration. The obliteration of single branches of the pulmonary artery in tuberculosis, the occlusion of these vessels so often observed in cases of extensive pneumonia, and other similar conditions, distinctly point to a secondary coagulation of a column of blood arrested by some mechanical obstacle. The primary occurrence of older clots (fibrin plugs), formed a long time before death, in the pulmonary artery, where it can be demonstrated that the obstruction of the artery precedes the changes in the parenchyma or takes place independently of them, is always secondary as regards the site of the clot; *i. e.*, these plugs originate in some part of the vascular system situated back of the lungs, in the veins or in the right heart, and are carried into the pulmonary artery by the blood-current ([212], page 224).

The extent of the subsequent local disturbances in the lungs depends partly upon the occlusion itself, but chiefly upon the character of the occluding body. An experiment on a dog in which three rubber plugs were used as embolic material, the animal being killed after three months, showed that the affected part, the left lower lobe, was perfectly intact, although the supplying branches of the pulmonary artery were made absolutely impassable by the rubber plugs. The nutrition of the lungs had been sufficiently kept up by the much dilated bronchial artery and by the intercostal arteries communicating with it. Material injected into the aorta entered the pulmonary lobe through these vessels ([212], page 295).

The thrombi which form within the lungs by secondary coagulation around the embolic plugs may either become organized into a vascular, cellular connective tissue, in which case the colorless blood-corpuscles are converted into connective-tissue corpuscles, or break down into detritus, either by simple softening or by putrid deliquescence. Simple or putrid detritus formation was found in most experiments in which elder-marrow and organic substances were used as embolic material.

Atrophy of the pulmonary tissue, pneumonia, pulmonary gangrene, and hemorrhagic infarct are discussed as possible consequences of embolism.

These investigations found a valuable confirmation in the numerous experiments and clinical observations of Cohn and Panum. Their investigations also prove, among other things, that the non-appearance of infarct after occlusion of an arterial branch depends on the properties of the embolus. An important observation by Panum shows that immediate occlusion of the pulmonary artery by large and small plugs, like balls of wax, for instance, which do not materially irritate the pulmonary tissue either mechanically or chemically, has no other effect than the encapsulation of the plug at the place where it becomes arrested, and does not give rise to any other nutritive disturbances in the pulmonary tissue. On the other hand, a perfectly fresh clot of healthy blood may, if it forms an embolus in the pulmonary artery, produce typical lobular processes or pulmonary infarcts in the same individual from whom the blood-clot is derived. But it is only under special circumstances that they have this local effect; the majority of them shrink and dissolve without producing any material change in the neighborhood of the place where they are situated.

Cohnheim bases his investigations of embolic processes in the lungs on the dendritic division of the pulmonary artery and on the absence of anastomoses except between the last arterial twigs, that is, within the lobular septa. Therefore if an embolus gets into a twig of a certain caliber, a hemorrhagic infarct is produced behind the embolus. The formation of the latter he attributes, on the strength of his experiments on a frog's tongue, to a retrograde movement of the blood in the valveless veins which belong to the occluded artery. This leads to engorgement of the vessels, and later on,—after some hours, in consequence of the changes that have meanwhile occurred in the vessels of the embolized district, especially the capillaries, in consequence of the interruption of the regular blood-supply,—to the extravasation of the blood into the tissue, in other words, the infarct.

Cohnheim contends that this is the only possible explanation of the fact that the infarcts arise some time later instead of immediately after the embolism of the artery. This fact was bound to appear enigmatic, as long as it was explained by the collateral arterial flow, since the force of the blood-current must necessarily be greatest immediately after the occlusion, and any danger from that quarter must diminish as the process continues. His interpretation, he claims, also explains the fact that in a hemorrhagic pulmonary infarct the arterial embolus does not by any means occupy exactly the apex of the reddish-black wedge, or infarct, but is always found slightly to one side of the apex, because the territory occupied by the infarct corresponds directly to the territory of the pulmonary vein, and only indirectly to the distribution of the artery; for the two systems do not run close together in the lungs, their branches being always separated by pulmonary tissue.

In regard to the formation of abscesses in consequence of embolism, Cohnheim points out that infarcts are always situated at the periphery and have an approximately conical shape, so that the base of the infarct is situated at the periphery of the lung,—*i. e.*, in the pleura,—and the apex is directed toward the hilum; whereas abscesses are not at all limited in their localization to the periphery, but occur anywhere in the lungs, even quite near the hilum, and very frequently at a distance of one or two centimeters from the pleura. Furthermore, abscesses are always practically spherical, so that even those which extend to the periphery do not have their largest diameter at that point, but touch the pleura only with a comparatively small segment of their convex surface. An infarct is the result of the occlusion of an end-artery. An abscess results when a vessel other than an end-artery becomes occluded by an infected plug. It is because the vascular arrangement of the lungs is such that an embolus which has entered the pulmonary artery is just as likely to reach an end-artery as one possessing anastomoses further on, that this organ is particularly adapted for the typical development of either mode (page 413) of termination above described.

Cohnheim's explanation of the processes in hemorrhagic infarct was destined soon to be shaken to its foundation by further investigations of the vascular relations in the lungs. Küttner proved that the vascular territory of the pulmonary artery is by no means as isolated as was contended, and that the separate twigs communicate extensively with one another, although only by means of capillaries; while ample relations exist between the territory of the pulmonary artery and that of bronchial arteries, a fact which is the more significant as the twigs of the latter are mutually connected by arterial branches. He did not succeed, however, in proving anatomically the existence of preformed communications other than by capillaries between the branches of the pulmonary artery. Additional connections can, however, undoubtedly be established, and render feasible an abundant flow of blood from one vascular district into the other, if the anatomic configuration of the vascular tract of the pulmonary artery, its relations to the vascular area of the bronchial artery, and the results of experimental investigations are taken into consideration. The most important result of these investigations is that cinnabar injected into the blood of animals after ligation of the pulmonary artery is found not only in the branches of the bronchial artery, but also in the branches, as well as in the trunk of the ligated pulmonary artery, in the capillaries of the alveolar walls, and in the pulmonary veins.

After ligation of the common trunk of the pulmonary artery of the left lung the intrapulmonary trunk of this vessel, as well as its larger branches, were found to be filled with a coagulum of blood and fibrin, and in the lungs proper marked congestion and even hemorrhagic infarcts were produced in a short time.

Litten positively contradicts Cohnheim's assumption that the infarct is brought about by a backward flow of the blood in the venous district corresponding to the infarcted artery. He maintains that the backward venous flow after ligation of the pulmonary artery is not by any means essential to the production of an infarct; for he found that when both the pulmonary artery and the veins were tied, precluding the possibility of a venous engorgement, a typical infarction constantly followed. But this hyperemia and infarction do not occur if, in addition to the pulmonary artery, the bronchial artery and those arteries which are outside of the lungs, but communicate with the bronchial artery (arteriæ tracheo-œsophageæ, pericardiacophrenicæ, pleuræ-mediastinales), are at the same time excluded from the circulation. If the lungs are thus deprived of their entire arterial supply, and their sole vital connection with the remaining organism is through the veins, infarction does not follow, whereas it does occur immediately if, with open veins and ligated pulmonary artery, the flow of blood through the collateral arteries into the lungs is not interfered with.

Litten concludes from these facts that a venous reflux cannot set in as long as the circulation in the pulmonary capillaries is maintained by collateral arterial branches. If a hyperemia of the lungs, which may go on to hemorrhagic infarction, is a consequence of the ligation of the pulmonary artery, it is more correctly explained as follows: Under physiologic conditions—*i. e.*, with unimpeded circulation—the entire resistance to the blood-current in the capillaries of the lungs is overcome by the blood-pressure in the pulmonary artery, which, in keeping with the greater width of the capillary lumina, is much less than the pressure in the arteries of the greater circulation. If the artery suddenly becomes impassable, the pressure in the collateral arterioles, which are derived partly from the bronchial artery and partly from the above-mentioned arteries outside of the lungs, is strong enough to prevent a reflux of the venous current, but not sufficient to overcome the total resistance in the lungs and to force the blood through the capillaries into the left auricle. Thus accumulation and stagnation of blood in the capillaries and smaller veins are brought about; the latter become abnormally distended and are thus in the same condition as if the vein into which they empty were constricted. The result of this condition is at first hyperemia and later diapedesis.

The statements of Litten, being based on facts, are such as to cast a doubt on the correctness of Cohnheim's explanation of the mechanism of hemorrhagic infarcts; they actually do away with the necessity for assuming a backward flow in the venous branches accompanying the occluded artery. This assumption appears *à priori* somewhat forced. Considering the extremely small resistance, which under normal conditions is offered to the return flow of venous blood through the pulmonary vein to the left auricle, it is difficult to understand how a backward flow into the part affected by an embolus, resulting in a copious extravasation, can be brought about.

Before the discussion of the manner of production of an infarct can be carried any further, the question whether the pulmonary infarct is without exception the consequence of embolism of the pulmonary artery or one of its twigs must be decided. The most careful author on this subject, Luzzatto, in his great work on embolism of the pulmonary artery, arrived at the conclusion that hemorrhagic infarct in the lungs depends without exception on embolism of the pulmonary artery. He contends that the following facts are conclusive: (1) In almost all dissections of hemoptoic infarcts a coagulation of embolic origin is found in the respective arterial twig; (2) a similar kind of infarct can be produced experimentally; (3) it is scarcely comprehensible how else a lesion could be produced which is exclusively limited to a vascular area belonging to a single arterial twig. In thrombosis, which develops slowly, compensation may readily be brought about by the collateral circulation; and it is hardly conceivable that the effects of stasis should be limited to a definite portion of the pulmonary tissue supplied by a single arterial twig.

This argument, however, does not appear to me to explain the fact that infarcts are comparatively frequent in valvular disease of the heart, as well as in affections of the myocardium, although, in spite of careful search, it is not rarely impossible to discover either in the right heart or in the peripheral veins any changes that might demonstrate the embolic character of the infarct. The majority of authors, therefore, believe that infarcts may occur without embolism, especially in the presence of valvular lesions and protracted conditions of myocardial weakness.

Even at present, however, two different views are held in regard to the occurrence of infarcts *without preceding embolism.* According to one view, the infarct is produced by a laceration of vessels, as Laennec and Rokitansky long ago contended. Friedreich attributes the production of hemorrhagic infarct in heart disease to the laceration of larger vascular branches, and, with Dittrich, considers fatty degeneration of the walls of the pulmonary vessels, which frequently occurs in consequence of increased pressure in the lesser circulation, a favorable condition for this rupture. Rindfleisch even says that the infarcts which occur in consequence of embolism of larger branches of the pulmonary artery differ from those depending upon laceration of a large vessel, in so far as in the former the transition from the infarcted parenchyma to the surrounding normal tissue is very gradual, presenting every phase from a hemorrhagic hyperemia to simple hyperemia, while in the latter the infarcted portion represents a firm, uniformly dense, dark blood-red wedge, abruptly separated from the healthy tissue. This kind of hemorrhagic wedge, according to Rindfleisch, is not brought about by embolism, but by rupture of a large vessel, and is mostly found in individuals suffering from heart disease. The increase of the lateral pressure in the lesser circulation must be regarded as the predisposing cause. In this respect the infarct represents a quantitative excess of those capillary hemorrhages which occur in brown indurations. The immediate cause which principally determines the location of a vascular rupture, is probably to be sought in fatty degeneration of the medium-sized and smaller branches of the pulmonary artery; while a passing congestion in the lungs represents the exciting cause which brings about a collision between the diminished resistance of the vessels and the increased demand upon them. Ziegler also favored the opinion that when the heart action is weak, or valvular defects are present in the left heart, causing stagnation of blood in the pulmonary vessels, the affected vascular area becomes filled with stagnating blood, so that extravasations take place which lead to the formation of a hemorrhagic infarct—*i. e.*, to a firm infiltration of the pulmonary tissue with coagulating blood.

According to my experience, the assumption is not tenable that infarcts are the result of extravasation in consequence of laceration of vessels. In the first place, there is no positive proof, and the theory fails to explain satisfactorily the sharp limitation, the characteristic form, and uniform density of the infarct, in both the periphery and at the center. Furthermore, the shape of all infarcts is the same, whether their embolic nature can be demonstrated or not. Finally, it is to be observed that in those conditions which, without presenting any evidence of previous embolism, are followed by infarction—that is to say, in heart disease—the occurrence of profuse pulmonary hemorrhage is extremely rare (I am almost inclined to say unknown) in comparison with the frequency of infarcts, notwithstanding numerous clinical and anatomic observations have been reported—another argument that the transition from hemorrhage to infarction is improbable. On the contrary, experience shows that the infarct appears first and is followed, at the earliest, after several hours by the expectoration of blood.

Besides pulmonary hemorrhage, which is not tenable as a cause of infarct-formation, **local thrombosis** has been considered as an etiologic factor. The effect of thrombosis, it was said, must be the same as that of an embolus derived from a distant portion of the circulation; hence demarcation, form, and consistency of the infarct must be the same; but there was no proof to show that such a process takes place.

Lately Grawitz very emphatically maintained that local thrombus formation in pulmonary vessels is by far the most important factor in the production of pulmonary infarct. He bases his opinion on the fact that hemorrhagic infarct is never met in a normal portion of the lungs, severe circulatory disturbances being apparently necessary to prepare the way for its formation. These disturbances not only include red and brown induration of the pulmonary tissue, but also certain sequels of bronchitis, such as partial atelectasis; also vascular cicatrices at the sharp pulmonary borders, both in the subpleural and in the interlobular connective tissue. He further explains that three different pathologic changes capable of promoting the formation of parietal thrombi are found in the arteries of the lungs: namely, (1) Hemorrhage between adventitia and media, more rarely, between media and intima. He considers this to be the most frequent cause of thrombosis. The hemorrhages he ascribes to a vascular peribronchitis which leads to venous engorgement and rupture of the newly formed bronchial vessels. The blood forces the adventitia of the neighboring vessels apart, advances to the media, and may in this manner suspend the circulation in the arteries entirely. (2) Pulmonary endarteritis or fatty degeneration of the intima may lead to thrombosis. (3) Chronic inflammations of the lymph-glands which are situated within the lungs, occupy the bifurcation of the bronchi, and appear to cause cell proliferation in the neighboring arterial walls, are among the possible causes.

Grawitz comes to the conclusion that many hemorrhagic infarcts do not contain any clots in the supplying arteries at all, and that a great many of the plugs which are found in infarcts are not emboli, but thrombi which are either continuous with the infarct or originate in diseased places in the arterial wall.

Willgerodt, Orth, and Fujinami have tried to disprove the assertion of Grawitz, who accords such a prominent part in the production of infarcts to thrombotic processes; Willgerodt by appealing to numerous anatomic specimens, the other two by repeating, with certain modifications, former experimental attempts to produce embolic processes. Fujinami also states that hemorrhagic infarct may be caused by an embolus alone in absolutely healthy lungs, "if all the conditions were complied with," but that, at the same time, other points must be taken into account. In the first place, the hemorrhagic infarct generally occurs in adults who have suffered from chronic affections of the lungs in consequence of which the pulmonary circulation has naturally become impaired. Second, embolism goes hand in hand with other affections which have an injurious influence upon the circulation in the lungs and undoubtedly may take part as auxiliary factors in the formation of the hemorrhagic infarct.

It must be especially mentioned that lately Gsell has achieved much more favorable results than former authors, by introducing paraffin into rabbits by way of the jugular vein. He succeeded in producing, without exception, in connection with the paraffin embolism, changes of different grades in the embolized district up to a typical hemorrhagic infarct. He believes that this difference in the results is due to the fact that others took paraffin of higher melting-point and worked during colder weather than he did. An embolus with soft paraffin such as he used, principally during summer, caused not only a simple occlusion of the arterial branch which was entered by the large plug, but also obstruction of its finer twigs. From this he deduced that the combination of large and small emboli in the same district is bound to have graver consequences than the occlusion of a larger branch alone. He considers the soft paraffin to be especially effective for still another reason. The plastic properties of the plugs allow them to stretch and thus occlude a long section of an artery with lateral twigs, and this is a potent factor in preventing the establishment of an adequate collateral circulation.

For my part, my own clinical and anatomic observations lead me to believe that the great majority of pulmonary infarcts are due to the obstruction of smaller arteries by emboli, which are unquestionably more effective—as will be shown later on—than elder-tree marrow and paraffin, the materials used generally for experimental obstruction of pulmonary vessels; but I am now in the possession of positive findings which prove that a limited number of pulmonary infarcts, especially of those which occur in heart disease, must be ascribed to a local thrombosis.

In September, 1897, Mrs. E., thirty-one years of age, was admitted to the hospital on account of a grave disease of the heart which set in after rheumatism and had existed for four years. She died on October 18th of the same year. Twenty days before death she had a slight stitch in her left side which soon passed off, and eight days before death she had another severe attack of pain in the same side which lasted several days. These pains were complicated by the appearance of blood streaks in the sputum, and five days before death the sputa contained more abundant bright red blood. The autopsy showed a stenosis of the aortic valves and an insufficiency of the mitral valve; in the left lower lobe an infarct the size of a walnut was found.

The microscopic examination of this infarct showed certain changes which were quite noteworthy and help to explain its origin. Instead of blood being found in the alveoli, as was to be expected, a considerable number of them contained swollen alveolar epithelium. Other alveoli contained swollen epithelial cells, elements of more than normal thickness without nuclei, and, in addition, some intact red blood-corpuscles. Between all these formed elements a crumbling mass was observed which, judging from its color, might have been produced by the disintegration of red blood-corpuscles.

It is remarkable that such a composition of the infarct has so far been described by only one author, Heschl. He remarks that, in addition to hemorrhagic infarcts, areas of lobular inflammation are also found, from one to four in number, and not less than pea- nor more than walnut-sized, in which the epithelial cells are enlarged, mostly transformed into granule cells, and partly of a yellowish-brown color. Pus-corpuscles may possibly be found as well. He saw practically the same picture in many infarcts, except that the vessels were also distended with blood. He concluded from this that the infarct represents the second, more advanced stage of the lobular infiltration.

In my case, however, not only the alveolar contents, but also the blood-vessels leading into the infarct, showed important peculiarities. This vessel was 3.2 mm. wide, and even macroscopically it could be seen that it was filled with a clot. The microscopic examination of cross-sections of the vessel showed that about two-thirds of the periphery of the clot was adherent to the vessel-wall, while the remaining third was free, leaving an empty, crescent-shaped space which measured 0.6 mm. at its widest place. At the place where the clot was adherent to the vessel-wall a single layer of remarkably large round cells with large nuclei was interposed. Similar cells were found scattered in the upper layer of the intima. Within the single layer of round cells some intact red blood-corpuscles were also found. All the remaining solid contents consisted of nothing but red blood-corpuscles, part of which were intact, while others were pale, but most of them had coalesced so that their boundaries were indistinguishable. They were so close together that only here and there fine threads of fibrin were visible. But that part of the entire local process which I consider most important I found in the vessel-wall itself. As will be seen in figure 13 on Plate 7, the adventitia is enormously increased in width, making up 1.4 of the 2.6 mm. which represent the entire width of the vessel-wall. At the same time, the adventitia consists almost entirely of heterogeneous fragments without form or structure, and mostly oblong in shape. But this change was found not only in the part shown in the illustration, which corresponds to the seat of the occluding mass, but also in finer arterial twigs, the lumen of which was empty. Everywhere this structureless transformation occupied at least half of the entire thickness of the vessel.

Here we are dealing with a change of the adventitia which agrees with the "hyaline degeneration," so called by Recklinghausen, and with the process which Neumann calls "fibrinoid degeneration." Regarding the manner in which this change is produced opinions still differ. For my part, the observations in this case, and the transformation of the connective tissue into oblong, hyalin-like, structureless, broad bands, described under Chronic Pneumonia (page 694), lead me to indorse Neumann's view, that "the complete absorption of the fibrillary structure and the formation of an absolutely homogeneous, structureless, hyaline mass speak much more in favor of a change occurring in the tissue fibers proper than for a simple saturation of the tissue with a coagulating exudate," or, as I should like to call it briefly, a *swelling of the tissue elements* which in this case partake of the character of connective tissue. But this swelling may also affect other elements, for instance, the membranæ propriæ of the kidneys.

In the variety of infarct here under discussion there can scarcely be any doubt that the fibrinoid degeneration of the adventitia takes place before the changes in the interior of the vessel, and that consequently, in the absence of any evidence of the embolic nature of the process, and in view of the shape of the clot, which does not quite fill the lumen of the vessel, we are dealing with a local thrombosis which is quite properly referred to the disease of the adventitia.

This connection is made still more probable by the fact that such a change in the adventitia is apparently inexplicable on the theory of a primary process within or on the inner surface of the vessel-wall; furthermore, it can be proved by the clinical observation of this case that infarction takes place during the last days of life, whereas a profound fibrinoid degeneration would require a longer time for its development. Finally, the priority of the fibrinoid degeneration can be proved by the fact that the smaller vessels within the infarct had undergone the same alteration of the adventitia, although their lumen was absolutely empty.

For these reasons the fibrinoid degeneration of the adventitia of smaller pulmonary arteries may be considered to be an important etiologic factor in the production of thrombosis, which, in its turn, is followed by the formation of an infarct.

Since this first observation I have seen only one case of infarction in the train of an aneurysm of the aorta in which every trace of an embolic origin was absent. This case was that of a man, forty-seven years of age, who had been infected with syphilis twenty-five years previously. He had always been healthy since then, and only six months before his admittance to the hospital, on July 2, 1898, was attacked by cough and dyspnea. The examination showed an increase of the cardiac dulness, a loud diastolic murmur over the heart, and slight retraction of the apex. Over the right lower lobe dulness, enfeebled respiration, and râles; on the left side anteriorly, above the heart, an empty high-pitched note and roughened breathing were present. Six days later the sputa consisted of pure blood, and the diagnosis of "infarct of the lungs" was made in consequence. Six weeks after admittance death followed. The autopsy showed: Considerable dilatation of the chambers of the heart, no affection of the valves, and aneurysmal dilatation of the ascending aorta in consequence of high-grade atheroma. While the circumference of the aorta just above the valves amounted to 7.3 cm., the measurement of the ascending aorta at its widest place was 10.5 cm. In the anterior middle portion of the left upper lobe a consolidated area as large as a hen's egg was found, which, on the cut surface, had a rosy appearance, and in a few places presented a yellowish discoloration (brown induration). The right pleural cavity contained 900 c.c. of reddish bloody fluid. At the anterior edge of the upper lobe, immediately below the surface, was a hazelnut-sized focus which consisted of a hard, solid piece of flesh-colored substance which was completely separated from the surrounding pulmonary tissue. It therefore occupied a cavity the walls of which were smooth and covered with a layer of pus. In addition, the posterior lower edge of the right lower lobe contained another wedge-shaped focus, half as large as a plum, which had a mottled, blackish and grayish-red appearance.

I was able to demonstrate the same fibrinoid degeneration of the adventitia as in the previously mentioned case, both in the older infarct, which was completely separate from the surrounding tissue, and in the recent one. The alveoli were almost filled with red, more or less plainly recognizable blood-corpuscles, among which only a few isolated cells of alveolar epithelium were visible.

However, it must be left to future exhaustive investigations to decide whether protracted affections of the heart or stasis in the lesser circulation, which may be due to other causes, such as aneurysm, for instance, in their future course regularly lead to fibrinoid degeneration of the adventitia, which itself may be considered the cause of local thrombosis and indirectly of infarction; and whether this degeneration occurs some time before the formation of the thrombus, *i. e.*, independently, in lungs which have become permanently injured by the stagnation of blood due to such disturbances.

Supported by the cited cases and the results of my own investigations, I may now answer the questions which were asked at the beginning of this section as follows:

There is no infarct of the lungs without embolism or without thrombosis. It is to be considered unproved that in a normal pulmonary tissue, or even in one which is pathologically changed by blood stagnation, simple rupture of a vessel may lead to the formation of an infarct. At least the existence of atheroma, which is said to precede such a laceration, in blood-vessels of small caliber, such as are affected in infarcts, would have to be much more positively demonstrated than has hitherto been the case. Furthermore, the hemorrhage which would take place into the surrounding tissue at the seat of rupture would not have the characteristic wedge-shape of an infarct and would involve a participation of the pulmonary tissue surrounding the infarct. But such participation is wanting.

In accordance with the majority of the authors named, I consider most pulmonary infarcts to be the result of **embolism.** Clinical observation even warrants the assertion that they are an immediate, direct consequence of embolism, and that the pulmonary tissue may have been perfectly intact up to the moment of occlusion of the respective vessel by the embolus. When a robust butcher, thirty-seven years of age, whom I have known for years as absolutely healthy, contracts a thrombosis of the veins of the leg, proved by edema of the foot, after an inflammation of the knee-joint following a severe contusion of a *mus articularis* that had been caught between the articular surfaces of the knee-joint, and eleven days after the contusion an infarct characterized by hemoptysis, intense pain, and dulness over a circumscribed area of the lungs develops, then there can be no doubt that an embolus from the thrombosed vein in the leg has entered a pulmonary artery, and that the affected pulmonary tissue was previously intact. I have minutely described this case elsewhere.

In the case of a previously healthy man, thirty-four years of age, who was attacked by paratyphlitis, later developed thrombosis of the right femoral vein, and finally had a pulmonary infarct with hemoptysis, I have no doubt whatever of the previous soundness of the lungs, particularly as I had the opportunity of observing this man for fourteen years after he recovered from the paratyphlitis and its consequences, both in social and in professional intercourse, and could never demonstrate a symptom of disease in him. Whether, however, the fatal attack of gangrene of the lungs which occurred after the lapse of so many years had any embolic connection with the previous paratyphlitis could not be decided, because autopsy was not permitted.

When a woman fifty-six years of age is attacked by a pulmonary infarct with hemoptysis, after having been admitted to the hospital for a large carcinoma of the gall-bladder; when at the autopsy in otherwise normal lungs the presence of the same is demonstrated and a particle of carcinomatous tissue of the proper size—recognized as such with the aid of the following microscopic examination—forms the embolus in the respective artery, there is no more doubt possible of a direct and immediate connection between embolus and infarct.

The experiments with animals, it is true, appear to contradict such a supposition. The introduction of bland emboli—for instance, balls of elder-tree marrow, and paraffin—was not followed by infarction. But, apart from the fact that Gsell obtained better results than his predecessors, it has to be taken into consideration that a venous thrombus which gets into the lungs, even if it does not carry any infectious bacterial matter, contains other substances which have a greater influence upon the formation of infarcts than experimentally introduced balls of elder-tree marrow and of paraffin. The possibility that the thrombus originating in peripheral veins contains substances which exert an unfavorable influence upon the peripheral parts of the embolized pulmonary vessel cannot be excluded. Klebs, by increasing the coagulability of the blood (injection of fibrin ferment) in connection with experimentally produced embolic occlusion of the vessels, succeeded in producing infarction which he attributed to the progressing thrombosis in the periphery of the embolus.

The third question, Is it possible that in the normal parenchyma of the lungs an infarct occurs in consequence of *local thrombosis?* I have to answer in the affirmative after the above communications. But I cannot confirm the greater frequency of the thrombotic, as compared with the embolic infarct which is upheld by Grawitz. According to my experience, I can rather express the opposite opinion. I must also leave it undecided how much pathogenetic importance for local thrombosis is to be ascribed to the behavior of the pulmonary tissue, as described by Grawitz. I have not made any investigations on this point. Willgerodt and Fujinami have contradicted his explanations in some material points.

On the other hand, I can pronounce the fibrinoid degeneration of the adventitia of medium-sized arteries to be a cause of local thrombosis. But the consequences of this local thrombosis for the corresponding vascular district will coincide with the typical consequences of an embolism only when the thrombosis rapidly leads to complete occlusion of the vessel. Wherever this is not the case, an extravasation may not be the direct consequence. The tissue may instead experience mere inflammatory changes; at first a swelling and clouding of the alveolar epithelium sets in, as in the case described on page 733. Of course, a similar process may be consecutive to an embolus that only partially occludes the vessel, which then gradually becomes occluded by the deposition of coagulating blood.

The explanation of the **mode of production of infarct,** which was interrupted on page 730, may now be resumed. The former assumption that infarct is a consequence of arterial hyperemia and hemorrhage was only a makeshift. The sharp demarcation of the infarct from the almost normal surrounding tissue, the absence of such inflammatory changes in the later course of the process, the perfectly uniform infiltration of the tissue with blood within the affected vascular district, preclude a comparison of the process with one which takes place around a piece of tissue deprived of its blood-supply from the beginning; *i. e.*, necrotic tissue.

For a time, Cohnheim's theory that the origin of the infarct, especially the extravasation of blood into the territory corresponding to the occluded artery, is due to the backward flow in the veins, found many followers. But the experimental foundation for this explanation was shaken so completely by investigations of Litten and Küttner that the structure erected on the same could not hold.

On the strength of the investigations reported by Litten, Küttner, and Klebs, I feel inclined to attribute the origin of the infarct within the embolized or thrombosed vascular district to a hemorrhage from the local capillaries into the tissue, especially the alveoli. In the first place, the remarkable uniformity of the anatomic appearance of the entire infarct on section, even the granular surface which is described by some authors, indicates the capillary origin of the blood. But a far more important point of support is offered by the fact, confirmed by all investigators, that extensive communications exist among the capillary vessels of the pulmonary arteries, and that the capillaries of the bronchial arteries are in manifold communication with those of the pulmonary arteries. I could plainly convince myself of the connection between the capillaries of the bronchial arteries and those of the pulmonary arteries at the junctions of the finest bronchioles with the alveoli while inspecting preparations of influenza-pneumonias which, stained with the Biondi-Heidenhain mixture, give pictures as good as are obtained by artificial injection.

On this positive basis we may assume that the capillaries in the district supplied by the embolized or thrombosed artery suffer some injury which is caused partly by the anemia and partly by chemical processes induced by the occluding coagulum. In consequence of this injury the capillaries in the affected district are not able to transmit the blood which flows into them from the communicating healthy capillaries; they tear, and thereby the uniform infarction, or the extravasation of blood into the alveoli, and furthermore the expectoration of blood, are accomplished. Not without importance for this process is the fact also that by the absence of the arterial filling of the entire embolized district a decrease in the tension of the entire tissue is produced from the very beginning which assists the lacerating tendency of the tissue.

With regard to the **seat of the infarct,** the frequency of its occurrence at the periphery of the lungs, especially in the lower posterior edge, must be emphasized. The base of the infarct, which is usually wedge-shaped, is then found immediately under the pleura. One part of its base faces the costal, the other the diaphragmatic surface. But it may happen that the infarct is spheroidal, and part of its convex surface bulges toward the costal, the other part toward the diaphragmatic surface of the pleura, so that just the edge of the lower lobe remains intact. Infarcts situated in the interior of the lungs are also spheroidal in shape. Their occurrence in man has been proved beyond doubt. Here Cohnheim's dictum, based on experiment, that "the seat of the infarcts is always peripheral," does not apply.

Emboli usually get into the lower lobes, and unless special causes change their direction are more frequent in the right than in the left lung, as the blood-current is stronger on that side. This is confirmed by all statistics (Gerhardt). The following are considered to be the conditions for embolism and infarction on the left side: (1) Weakening of the current in the right pulmonary artery by previous multiple or large emboli on the right side, or by right-sided thrombosis of the pulmonary vein (Penzoldt); (2) weakening of the current by shrinking of the right lung, compression by pleural exudates, and similar causes (Gerhardt); (3) increased flow of blood into the left pulmonary artery in consequence of stronger respiratory movements of the left side when the individual lies on the right side (Penzoldt).

The pathologic **termination** of infarct, if the process lasts long enough, manifests itself in atrophy of the affected part and transformation into a fibrous mass. The processes which lead to this condition consist at first in disintegration of the red blood-corpuscles which are deposited in the alveoli in a granular and crumbling mass. But some red blood-corpuscles continue in the alveoli for some time, although they are discernible as such only by their form and size, the pigment having disappeared. The capillaries of the alveolar walls very soon disappear from view, probably because they become empty and collapse. The alveolar walls proper appear widened; their structure cannot be distinguished through the deposited granular masses. Sometimes varying quantities of alveolar epithelial cells are found in the alveoli, but their nuclei have entirely disappeared. Sometimes fibrin threads are seen, but rarely in any considerable quantity.

Another termination of the infarct is by complete separation from the surrounding healthy pulmonary tissue, as has been described in the case reported on page 735. The differences between this process and embolic abscess consist in the uniformly firm consistency and blood-red color of the infarct which is cast off as a whole. We shall more explicitly dwell upon these processes in the sections dealing with abscess and gangrene of the lungs.

ETIOLOGY.

After proving in the preceding investigation of the pathogenesis of the disease that infarct of the lungs is only the consequence of occlusion of the vessel which supplies the infarcted part, and that the occlusion may be caused either by an embolus or by a local thrombosis, it will suffice in treating of the etiology to discuss the *origin of the embolus* and the *cause of the local thrombosis.* Consequently two etiologic groups are to be distinguished from the beginning.

To the *first group* belong all solid bodies which are formed by pathologic processes in any portion of the venous system of the body, in the right auricle, in the right ventricle, in the trunk and in the main branches of the pulmonary artery; or penetrate into the lumen of these channels and spaces which otherwise only carry liquid blood. The former embrace coagula originating from pathologic changes in the intima of the veins of the greater circulation, in the endocardium of the right heart, or in the intima of the pulmonary artery; the latter include neoplasms that have pushed through the vessel-wall into the lumen, or into the cavity of the right heart, and tumor particles that have been carried into the lungs by the venous blood-current. Particles of carcinoma and sarcoma are the commonest of these pathologic products.

If I may name the processes which lead to pulmonary embolism according to the frequency of their occurrence, the formation of thrombi within the venous system must be named in the first place, and at the head of the list is *thrombosis of the veins of the leg.* It occurs most frequently in the course of protracted febrile diseases that are accompanied by considerable anemia. According to my experience, it is not much less rare as a consequence of trauma, *i. e.*, the laceration of some of the fibers of the muscles of the calf. Undoubtedly in consequence of this laceration an inflammation of smaller veins sets in, and thus brings about very favorable conditions for the deposition of blood on the intima. In the thrombosis which follows a fracture, the accompanying laceration of muscles is probably chiefly responsible for the origin of the thrombosis. The above-mentioned case, in which inflammation of the knee-joint followed a contusion of a *mus articularis* in a robust butcher, also belongs to the realm of traumatic phlebitis with thrombosis. Thrombosis may also occur in chlorotic individuals. In a recent work on thrombosis in chlorotics Schweitzer has demonstated that in 51 cases of thrombosis the thrombus was situated 33 times in the veins of the leg.

Next in the order of frequency are those emboli which originate in thrombosis of a vein after gynecologic operation, or in thrombosis of a vein in the neighborhood of the puerperal uterus. The latter cause would be observed much more frequently were it not for the fact that many of the emboli originating in this way carry infectious material and produce, instead of simple pulmonary infarcts, pyemic or gangrenous foci.

Thrombosis with consecutive pulmonary embolism as a consequence of surgical operations may now be regarded as a rare accident; a few isolated cases of thrombosis with consecutive embolism after paratyphlitis have been reported.

If an embolus of thrombotic origin gets into the lungs from other venous regions, principally from the sinuses of the brain, it mostly contains infectious material; hence in this case also simple infarcts rarely occur, oftener purulent or gangrenous processes in the lungs.

Among other sources of emboli are to be considered the inner surface of the right heart, and that of the pulmonary artery with its main branches. From the latter also the thrombus has to be carried for some distance before it is able to lead to an occlusion of vessels.

Embolism due to small particles derived from tumors that have grown into the vascular channel represents an absolutely analogous process so far as the formation of the pulmonary infarct is concerned. Only the tumor elements of the embolus increase at the point where it becomes arrested, and they grow through the vessel-wall, while the changes in the infarct take their own course as described above.

In regard to the *second group*, it appears that the occurrence of *thrombotic processes* in the pulmonary arteries presupposes some weakness on the part of the heart, such as valvular endocarditis, myocarditis, pericarditis, and atheroma of the aorta. At least I have observed the fibrinoid degeneration of the adventitia of the smaller pulmonary vessels, which I maintain is the direct cause of thrombosis, only after such affections.

It remains to be said in regard to the etiology that, while every infarct is the consequence of embolism or local thrombosis, every embolism and every thrombosis need not necessarily have an infarct in its train. This has special reference to very large emboli. They may occlude the main trunk or one of its lateral twigs, and cause death so rapidly that there is no opportunity for an infarct to form. Such observations have been made most frequently in gynecologic and obstetric practice. Wyder, Mahler, and others have reported a great number of such cases. Vice versa, embolisms may occur in which no infarct is formed on account of the smallness of the emboli. At least such a supposition is founded on clinical observations, as will be explained later on. But I would not venture to decide whether emboli of medium size, which only obstruct smaller pulmonary arteries, without exception produce an infarct in human beings, or whether some other conditions may not prevent the formation of an infarct in some cases. I only wish to express the supposition that in human beings every embolus of medium size possesses the chemical properties necessary to produce an infarct.

In local thrombosis of a small pulmonary artery the occurrence of an infarct depends principally upon the rapidity and the completeness of the obstruction of the vessel by the thrombus. The fact above referred to, that from the beginning, pneumonic changes, instead of an extravasation of blood, take place in the portion of lung supplied by the occluded artery, may have some connection with the gradual occlusion of the vessel.

SYMPTOMS AND DIAGNOSIS.

For the detailed description of the symptoms I shall adhere to the above classification of the emboli into *very large, medium-sized,* and *very small* ones. The symptoms which are caused by local thrombosis essentially correspond to those which are brought about by emboli of medium size. Large emboli which remain fast in one of the two main branches of the pulmonary artery, or in a large accessory branch, very rapidly cause grave clinical manifestations without leading to the formation of an infarct. The following description of the secondary manifestations of large emboli is given by Marfan; according to my experience, I can fully indorse the same:

Death may ensue so suddenly in the gravest cases of obstruction of the pulmonary artery by a **very large embolus** that the patient is not able to make any statement; but in most instances the affection commences with severe oppression and precordial distress, the patient is barely able to utter the words, "I choke, I die," drops, and is dead. It has not been fully ascertained how in such cases the cessation of the heart's action occurs. The sudden non-appearance of blood in the vascular system of an entire lung is undoubtedly of fundamental importance; the occurrence of a reflex paralysis, which may be in some way connected with it, must remain a hypothesis for the present.

In other cases *death* is not caused by syncope, but by *asphyxia.* The patient lives for a few hours or days. Such an attack always sets in suddenly. The patient has a violent sensation of suffocation, as though his thorax were constricted; the face, which at first is pale, speedily becomes cyanotic, the eyes actually protrude from their orbits, the pupils dilate, the jugular veins swell, the respiration is accelerated, the heart action becomes tumultuous and irregular. At the same time the patient complains of a continuous, severe, painful feeling of oppression; the want of breath is very great but cannot be satisfied; because although air enters the alveoli, the demand for oxygen cannot be satisfied because the required blood-supply is wanting.

Sometimes one observes torticollis, even general convulsions with frothing at the mouth, as in epilepsy. Death may follow in one to two hours, sometimes earlier. The pulse, which at first is very much excited, becomes feeble and intermittent; the patient has chills, his extremities become cold, the skin and the mucous membranes turn bluish. In other cases the fatal issue does not occur until after a day or two. The first oppression subsides and the patient rests more easily, but soon a fresh attack of dyspnea and oppression sets in, which may be repeated several times with remissions, until in one of these attacks life is extinguished. Consciousness is generally preserved, although sometimes delirium, vertigo, or convulsions may occur.

In some rare cases complete recovery may follow in spite of the described manifestations of intense asphyxia. But in most cases of that kind the diagnosis cannot be considered to be absolutely certain. I must leave it undecided whether this assertion does not apply to the following observation, which was made by me jointly with my colleague W.: A woman twenty-seven years of age, five times multipara, then in the second month of her sixth gestation, after lifting a heavy weight, on May 17, 1898, was attacked by a grave hemorrhage which was soon complicated by labor-like pains. After manual clearing out of the uterus the hemorrhage ceased and all troubles disappeared; only, in consequence of the preceding severe loss of blood, the slightly anemic, feeble woman felt very weak and faint. On June 1st (she had kept her bed without interruption) she was attacked by slight angina. During the night of June 4th suddenly high-graded dyspnea with cyanosis and severe oppression set in, the cause of which was ascribed to embolism, although nothing abnormal could be demonstrated by percussion or auscultation. After an injection of 2 cg. ($\frac{1}{3}$ grain) of morphin at 1.30 A. M. the patient's condition improved materially in the early morning, and toward evening all symptoms of dyspnea has disappeared. Gradual improvement followed; at the end of June she was perfectly well, and has continued to be so until now, the end of September (1898).

Recovery, as in this case, without the consecutive formation of an infarct, can be explained only on the supposition that the embolus has become arrested at the bifurcation of the pulmonary artery or at the point where one of its main branches is given off, and is astride of this bifurcation; so that only the first shock calls forth the severe symptoms, while from the beginning sufficient room is present in the lumen of the vessels to permit the circulation of blood, although in a limited measure.

There is practically only one process with similar termination that needs to be considered in the differential diagnosis—namely, the sudden occurrence of death in individuals apparently in the best of health from *atheroma of the coronary arteries*. The symptoms bear a great resemblance. The patients suddenly suffer from severe oppression, sometimes also from intense pains in the cardiac region. They have a presentiment of impending death. Very suddenly conspicuous paleness of the face sets in; unconsciousness follows; the pulse becomes feeble; and after a few labored respirations the fatal issue takes place.

Such an accident may occur without previous exertion on the part of the patient, or it may occur after pulling off the boots or straining at stool. If the history is carefully inquired into, it will be found that people who die suddenly in this way have frequently complained of pains in the cardiac region. The information that they have suddenly experienced a severe pressure in the cardiac region when walking, even on a level road, so that they were forced to stand still, is always significant. I have noticed this symptom in two men, between fifty and sixty years of age, during the last years before their death. The examination of the heart in these cases was absolutely negative; no enlargement of the heart dulness, no irregularity of the pulse, no sign of atheroma of the vessels. Both died suddenly with the symptoms described. The premonitory symptoms enabled me to make the diagnosis, and may do so in all cases of this character.

In the same manner as very large ones, **very small emboli** may enter correspondingly small vessels without causing infarcts. But I deduce this only from clinical observations. In two puerperal women, who had always been healthy, during the first days of the puerperium severe pains suddenly set in which were always located under the scapula at the level of the spine. The suddenness of the pain, its intensity, and its limitation to a small region presented a general picture which so completely corresponded to that of other emboli leading to infarct that I may assume the same process. The supposition is also supported by the predisposition which exists during the puerperium to thrombosis of periuterine veins, and also by the favorable result which followed a single injection of morphin.

Emboli of medium size most frequently lead to the formation of infarcts. They usually obstruct vessels 1 to 3 mm. in width. The symptoms of embolism in such a case are altogether absent or disappear very rapidly. They consist chiefly in sudden, intense pain at the point where later on the infarct can be demonstrated. If dyspnea is also present, it is usually due to the pain caused by the respiratory movements. It is rarely caused solely by the exclusion from the circulation of the affected vascular district. "Then it is possible that, with repeated chills, the respiratory frequency may increase to 60 and more per minute, and the anxious expression of the countenance, the lividity of the skin, the cold perspiration, complete the image of intense air hunger" (Penzoldt).

Cough may be insignificant in obstruction of pulmonary vessels by emboli originating in peripheral veins. In local thrombosis, with consecutive infarction, it depends more upon the fundamental cardiac affection, and does not necessarily experience an increase with the appearance of the infarct. But during the further course it may become materially aggravated. This is especially the case if the embolus carries infectious material, and a septic or gangrenous focus instead of a simple infarct develops.

Hemoptysis is the characteristic symptom of infarct. Pure blood is expectorated mostly in cases in which an embolus gets into a previously healthy lung from the peripheral veins. As the blood by means of the ciliated epithelium may be transported as far as the trachea and larynx, a single hawking movement frequently suffices to expel it. Coin-shaped masses of dark blood are then seen in a flat cuspidor. This is not always the case, however. More or less copious streaks of blood on the bronchial secretion may be all that can be seen, or the blood is so intimately mixed with the bronchial secretion that the latter assumes a dark red, gelatinous aspect. Sometimes, as has been mentioned by Laennec, expectoration of copious quantities of pure blood—*i. e.*, hemoptysis—occurs, and in these cases more or less severe cough will be present. The cough, at least at the onset of the infarction, is not so much a sign of the latter process as a symptom of bronchial catarrh which had been present from the beginning. Accordingly, it occurs in cases in which an affection of the heart is the fundamental cause.

The expectoration of blood does not set in immediately after the appearance of the embolus. Gerhardt, who observed spitting of blood 13 times in 15 cases, stated that it occurred once eight and a half hours, in two instances twenty-four hours, after the chill which marked the beginning of the process of embolism; and in other cases on the second or third day after the "paroxysm of suffocation."

The purity of the sanguineous sputum—*i. e.*, the absence of an admixture of bronchial secretion—is an important diagnostic point. The characteristic properties of pneumonic sputum, especially its toughness and rubiginous appearance, are rarely or never observed in hemorrhagic infarct. On the other hand, in croupous pneumonia the sputum may be almost purely sanguineous or very watery, or it may resemble prune-juice and contain only small quantities of pure blood, so that it will be very much like that of an infarct. In this case the physical examination, which in the case of pneumonia will show dulness mostly extending over an entire lobe, and the history will protect against error.

In an infarct the expectoration of sanguineous sputa lasts often for some time, even for several weeks. "They contain intact blood-corpuscles for more than a week. But after one to three weeks granules and crystals of hematoidin appear in the sputum, and after a variable time, often after one and a half to two weeks, the bloody sputa decrease and assume a brown, red, or blackish-red color, similar to that of currant jelly" (Gerhardt).

In most cases *percussion* yields trustworthy information in regard to the *seat* of the infarct, especially when the lungs were previously healthy and the embolus has originated in thrombosis of a vein. The diagnosis is more uncertain if thrombosis or embolism occurs in the course of a protracted stagnation of the blood in the lungs consecutive to cardiac disease. The stasis may previously have modified the percussion note, for instance, by brown induration, and it therefore becomes difficult to determine the seat of the infarct. Dulness may exist if the diameter of the infarct is but 4 cm., especially if its seat is peripheral (Wintrich, Penzoldt). In keeping with the above-described seat of infarcts the dulness is usually found in the right lower and in the middle lobe. The position of the patient, however, is not without influence upon the locality.

The *auscultatory signs* are usually restricted to crepitant râles over the dull area; bronchial respiration occurs rarely, "pectoral fremitus is increased" (Penzoldt).

According to my experience, *fever* does not occur in uncomplicated infarct. I fully indorse Gerhardt's words: "The temperature of the body, as a rule, remains normal; it may be below the normal, or show slight, irregular elevations."

Pleurisy is probably the only *complication* of infarct. It usually amounts to no more than a fibrinous coating of the base of the peripheral infarct; in some cases a large serous exudation may take place.

The appearance of pyemic and gangrenous foci bears the same relation to the embolus as the production of infarct; *i. e.*, if the embolus contains the bacterial causes of pyemia or gangrene, it must produce two different changes at the same time. On the one hand, by obstructing the artery it causes an infarct in the peripheral distribution of the vessel; and, on the other hand, it allows the pathogenic bacteria to develop.

The importance and the value of healthy organic tissue in the fight against bacteria are well established by general experience, and it goes without saying that the tissue of the infarct forms an excellent soil for the multiplication of the bacteria.

This division of the effect of an embolus into a local one, corresponding to the seat of the embolus in the pulmonary vessel, and a peripheral one, embracing the territory of the obstructed vessel, is best seen when the pulmonary embolus is a carcinomatous particle.

The growth of the cancer begins in the embolus proper; one can see how the nests of cancer cells permeate the wall of the vessel; the infarct is found at the periphery of the occluded portion of the vessel, and is in every respect similar to infarcts originating from other causes. But it is not *à priori* impossible that, although the embolus may not contain any pathogenic germs and therefore produces only a simple infarct, pathogenic germs may later on enter with the inspiratory air-current and colonize in the substance of the infarct. This is no doubt a very rare occurrence.

PROGNOSIS.

The prospects of recovery depend partly upon the size of the embolus, and partly upon the condition of the lungs at the time of the occlusion of the vessel. A third fundamental condition for the course, the presence of pathogenic bacteria in the embolus, need not be considered here, since it has been shown that the changes which they cause are independent of the infarct and belong to the realm of pyemia or that of gangrene of the lung.

Very large emboli which are retained in a main branch of the pulmonary artery may, as described, cause speedy death by syncope or asphyxia; termination in recovery is an exception.

While this kind of embolism has no infarct in its train on account of the rapidity of the fatal issue, very small emboli run a favorable course and do not produce infarction, because the embolized district is insignificant in extent.

Whenever a simple infarct develops, the prognosis depends essentially upon the condition of the lungs. If the latter have previously been healthy, the prognosis may be regarded as entirely favorable.

It is different, however, if in consequence of cardiac disease signs of congestion are present in the lungs. As soon as an infarct is formed, either by an embolus from the peripheral venous system or from the right heart, or by local thrombosis, the course depends chiefly on the existence or non-existence of compensation, or on the possibility of bringing it about by proper remedies. Even then the issue may often enough be favorable.

PROPHYLAXIS AND TREATMENT.

Many useful measures can be recommended for the **prevention** of embolic processes. As we know that emboli in the majority of cases are but detached pieces of thrombi which have developed in peripheral veins, and as such thrombi probably most frequently occur in the veins of the leg, where they can be demonstrated easily, the utmost should be done to prevent particles of thrombi from detaching. Even if only swelling of a leg occurs during an acute disease of long duration; or if a circumscribed painful swelling is present in the gastrocnemius muscle during such a disease, or without preceding disease after spraining of the muscles of the calf; or, still more, if on a swollen leg the femoral vein can be felt as a hard cord and is painful upon slight pressure, absolute rest in bed should be insisted on at once. Sometimes it is exceedingly difficult to enforce such a prescription. The director of an accident insurance company whom I had compelled to rest in bed quietly for several weeks, because he had contracted a thrombosis in consequence of overstretching the muscles of the calf, did not thank me until a year later, when his company had been legally forced to pay a considerable amount of insurance money to the heirs of a manufacturer who had also suffered from thrombosis consecutive to a distortion of the muscles of the calf, caused by stepping backward from a pile of iron pipes. On account of insufficient care the thrombosis led to fatal embolism of the lungs.

It is quite evident that *massage* of a leg swollen in consequence of thrombosis of the veins may cause the gravest danger, because pieces of the thrombus may thus become detached. It must not be applied under any consideration at first; and should not be practised for a long time, possibly months after the appearance of the thrombosis, when the thrombus has become completely organized and massage may be desirable to remove the remaining edema. I do not consider elevation of a swollen leg in thrombosis of any particular importance; it would better be omitted, especially as it is attended with a great deal of discomfort for the patient. Cold compresses, however, changed every hour or two and covered with a woolen cloth, can be recommended, but they should only be placed on the anterior surface of the leg, to avoid frequent raising of the same.

Not rarely in connection with the puerperium and gynecologic operations thromboses of the pelvic veins occur which may lead to embolism. According to Mahler and Wyder, a rapid pulse out of proportion to the temperature, which cannot be explained in any other manner, is indicative of the onset of thrombosis. "If a few days after a gynecologic operation, when the danger of sepsis has almost disappeared, and the patient is relatively or absolutely well, a sudden increase of the pulse is observed without any external palpable reason and with normal or nearly normal temperature, the phenomenon is of the greatest importance for the physician, because it indicates hidden or visible thrombosis, and if there is also edema of the lower extremities, which does not always depend upon a thrombotic process, is almost a certain sign that thrombosis is the cause of the swelling of the leg."

In all cases of this kind of thrombosis **iron** is indicated, and a **strengthening diet** can be advised if the condition of the stomach will permit it. Anemia of a more or less high degree is almost always present.

In the gradual development of thrombi in the right auricle or ventricle, in which sometimes also a small frequent pulse occurs, the prophylactic measures should be limited to what the prevailing condition of the heart demands. Rest, regulation of the heart action by digitalis, improvement of the blood by preparations of iron, are required in the first place.

Least accessible to therapeutic proceedings are local thromboses of smaller pulmonary vessels which appear in connection with cardiac defects and show fibrinoid degeneration, as described on page 734. The same applies to cases in which particles of neoplasms, which have grown into the venous or lymphatic system, detach themselves and gain access to the pulmonary circulation.

In the treatment of infarct the application of the various remedies depends primarily upon the size of the obstructing plug. If the emboli are very large, and unconsciousness, with or without convulsions, sets in almost at the same moment when the patient cries out that he has a feeling of impending catastrophe, the fatal issue can hardly ever be avoided. If immediate stoppage of the heart has occurred, neither faradization nor artificial respiration will be of any avail; but if consciousness remains, and if the embolism, by obstruction of a larger vessel, only leads to manifestations of grave dyspnea, I recommend with Gerhardt the subcutaneous administration of **morphin.** Neither cyanosis nor small frequent pulse must interfere with this procedure. All severe symptoms of disease disappear in some cases entirely, in others they are considerably alleviated, and thus the possibility of a uniform blood distribution is given with a hope of recovery. The subcutaneous injection of morphin should not be given in too small a dose. I recommend 2 cg. (⅓ grain) for adults, even if great weakness and anemia prevail, as in the case described on page 743. Very favorable and immediate results can be secured by the administration of morphin in patients in whom small emboli cause severe pain which increases the difficult respiration to dyspnea. In the above cases in which such manifestations occurred in the puerperium the effect of the injection was surprisingly prompt and lasting. The patients recovered at once, as no infarct followed the embolism.

Of course, even when the embolus causes infarct, and pains are the first symptoms of obstruction of a vessel, at first an anodyne, preferably morphin, may be required. But often the pains are slight or absent, and the first sign of embolism or thrombosis having set in is the evacuation of blood from the infarcted place. The quantity of the same is rarely large, but the expectoration of blood may continue for days. Therapeutically, hardly anything but **lead acetate** (every three hours, later three times a day, 3 to 5 cg.—grain ½ to j—with 5 dcg. —grains viij—of sugar in powders) can be recommended. Inhalations of the solution of the sesquichlorate of iron (liquor ferri sesquichlorati) or of other styptics I do not consider advisable on account of the deep inspiration required. **Opiates** or **morphin** against cough are indispensable. Rest in bed, bland but at the same time nourishing diet, care for regular, easy evacuation of the bowels, the avoidance of strong abdominal pressure, together with the remedies advised, should in most cases be sufficient to accomplish a cure. For, as long as no specific pathogenic bacteria combine with the embolus, consequently as long as neither abscess formation nor gangrene set in with, or, rather, locally in front of the infarct, complete recovery may be counted on.

Neither are local thrombi in the pulmonary tissue with secondary infarction in themselves dangerous to life, but only the affections which are the cause of thrombosis, such as valvular lesions and aneurysms of the aorta.

Even pleural effusion will interfere but little with the prospect of recovery. But it is better to leave the removal of the exudate to natural resorption and not to attempt its removal by puncture, at least not until the amount of the exudate and the dyspnea depending upon it urgently demand it, and then not to such a degree that the positive pressure which exists in a large exudate approaches the normal negative one, because this may cause a rupture of peripherally located infarcts.

ABSCESS OF THE LUNGS.

PATHOGENESIS AND PATHOLOGIC ANATOMY.

Abscess of the lungs, like the corresponding alteration in other parts of the body, means the infiltration of a piece of tissue of variable size with pus or white blood-corpuscles followed by severance of its organic connection with the surrounding tissue. The separation is probably brought about by the activity of the white blood-corpuscles alone without the co-operation of coagulation processes in the corresponding vessels. However, abscess of the lungs frequently differs from abscess elsewhere in the body in that the parts surrounding the tissue to be expelled are usually normal in other organs, while in the lungs a large portion, sometimes an entire lobe, may be the seat of some other affection, the abscess being formed later within the diseased part.

We therefore have to distinguish between abscesses occurring in *inflamed tissue*, and those that develop in previously *healthy lungs.*

Examples of the first variety are the abscesses which sometimes occur in *genuine pneumonia* and those which appear in the train of desquamative pneumonia (page 638). The process is easily explained by the above-mentioned definition of the nature of abscess formation. If in genuine or desquamative pneumonia the exudation of leucocytes at certain points or in a single circumscribed area is not confined to the alveoli, but also affects the interalveolar tissue, total softening of the loosened tissue, in other words, an abscess, results.

It is evident that the inflammatory process which, although originally more extensive, starts in the alveolar epithelium and runs its course within the alveoli, is the predisposing cause of abscess formation. In general, croupous pneumonia rarely terminates in abscess; but, according to the statistical investigation of Tuffier, this disease furnishes a larger contingent of pulmonary abscesses than any other. Of 49 operated cases of pulmonary abscess, 23 could be traced to fibrinous pneumonia.

All other forms of pulmonary abscess have the common property of developing in previously normal pulmonary tissue.

Abscess in consequence of *embolism* should be the first one to go into this class. Whether the embolus causes the formation of an infarct or not is a matter entirely independent of the formation of abscess. The abscess arises if infectious material reaches the lungs with the embolus from its place of origin, and the abscess forms at the seat of the embolus, not behind it, as does the infarct.

In such a case **multiple abscesses** instead of a single one are often present in the lungs, and with this is combined the clinical picture of pyemia.

Less frequently abscess of the lungs is brought about by the *aspiration of foreign bodies.* Whenever these are of a large caliber, and stick fast in one of the larger bronchi, extensive ulceration with the destruction of a large part of a pulmonary lobe is the result. In the case reported by Schild from the Magdeburg-Altstadt Hospital, the patient in trying to whistle on an acorn-shell, which he held between two fingers, aspirated the latter into the respiratory tract. Six days later resection had to be done for left-sided empyema, and $\frac{1}{4}$ liter of pus was evacuated. The resection wound closed very soon. The acorn-shell was coughed up six weeks after the accident, and nine months later the patient was discharged, but with manifest destruction of tissue in the lower half of the left upper lobe and in the lower lobe. Not until a year and three months after the onset of the affection, in spite of regularly repeated examinations,—the patient had remained under my permanent observation,—were tubercle bacilli found in the sputum, and six months after this he died.

Abscess following the *aspiration of fluids* or of very small corpuscular elements which enter the alveoli of the lungs is rare; as in such a case gangrene is much more common, unless death occurs before its full development.

After *injuries* which affect the lungs in the form of open wounds through the thoracic wall (perforating wounds of the chest) simple abscess is formed but rarely.

More frequent are the **perforating abscesses,** as they were called by Stokes. He applies this name to cases in which pus accumulates outside of the lungs, later penetrates the pulmonary tissue, and is evacuated through the bronchi. The starting-point of such abscesses may be the outer wall of the thorax and of the abdominal organs, an accumulation of pus in the pleural or in the peritoneal cavity, abscess of the liver (Stokes), a carious vertebra, suppurative bronchial glands (Leyden), echinococci of the liver, which will be expectorated with the sputum, or even a gastric ulcer, as I have observed in one case.[6]

Leyden, following Traube and Cotton, designates as **chronic abscess of the lungs** that form of simple necrosis and non-purulent ulceration (non-tuberculous) which most generally develops in the indurated lungs of the aged. The disintegration which is here observed in the lungs simulates in a certain manner the necrotic decay of indurated or slaty bronchial glands. As the process in the pulmonary tissue is quite similar to the insidious development of chronic abscesses in bronchial glands, which may even perforate the bronchus, the term chronic abscess appears equally justifiable in the case of the lungs. At any rate, this name is supposed to designate a chronic, simple—*i. e.*, neither tuberculous nor gangrenous—ulcer of the lungs, an ulcerating cavity filled with pus, but not a bronchiectasis, which has formed in consequence of necrotic decay of pulmonary tissue, that has been changed by chronic inflammation, and which progresses in the same manner. But, according to my opinion, it is further necessary to investigate whether some of these cases do not represent a sequestrum instead of an abscess. At least I should consider the possibility of Fraenkel's specimen, which he demonstrated as chronic abscess of the lungs, and in which a sequestrum as large as a hen's egg protruded into a large cavity in the right upper lobe, being really a sequestrum, and I would not admit a conformity with the pathologic process which occurs in abscess of the lungs, as described at the beginning of this section and also on page 486. But in every instance the co-operation of bacteria is of essential importance for the origin of abscess in the pulmonary tissue, whether they appear in infiltrated pneumonic parts or in a healthy organ.

Bacteriology.—Cohn, in an examination made with all proper precautions, found the pneumonia bacillus of Friedländer in the expectorated particles of tissue from a pulmonary abscess which had commenced under the clinical picture of pneumonia of the right apex and was cured later on, and pronounced it to be the cause of abscess formation. Zenker found in his case of abscess formation in pneumonia Diplococcus pneumoniæ as the sole cause of suppuration. In this case he explains the breaking-down of a poorly nourished pulmonary tissue, weakened by marasmus and an old influenza infection, into pus by the existing intense emphysema and the presence of enormous numbers of Fraenkel's Diplococci pneumoniæ, the virulence of which was probably increased by symbiosis with the influenza bacillus.

Koranyi demonstrated Staphylococcus pyogenes aureus by plate cultures in the blood of a patient, although no pathogenic bacteria could be demonstrated in the sputum. Pain prevailed in several joints, and infiltrations, first of the right, then of the left lower lobe had occurred. On the twentieth day of the affection 150 gm. of pus were expectorated; later, elastic fibers and shreds of parenchyma up to 2 cm. in length, and hematoidin crystals of various forms were found in the sputum. The woman recovered.

Hitzig attributes an abscess in the right lower lobe, in which the sputum contained elastic fibers, alveolar epithelium, and crystals of hematoidin to the direct action of the influenza bacillus. He made eight bacteriologic examinations of the sputum within five weeks, and in every instance he found typical influenza bacilli, but never streptococci, staphylococci, or capsulated diplococci. The patient, a woman fifty-five years of age, recovered.

It must be concluded from these results that various kinds of bacteria may be the cause of pulmonary abscess. It is reserved for future investigators to demonstrate what variations in the form, in the anatomic behavior, and in the clinical course of pulmonary abscess depend upon the different kinds of bacteria.

SYMPTOMS.

The presence of an abscess of the lungs can be positively assumed only when *elastic fibers* can be demonstrated in the sputum. Sometimes shreds of tissue several centimeters long are found, as Traube has shown (see page 481). Otherwise the microscopic appearance of the sputum does not present anything characteristic; during the greater part of the disease it corresponds to the affection which has led to the formation of abscess. If it was a pneumonia, however, the sputum as a rule soon begins to differ from that of typical pneumonia; according to Leyden, it is hemorrhagic or blackish; it may also be grass green, as found by Traube. Leyden describes the odor of the pus on the whole as musty. It may become offensively sweet, or even fetid, but these changes are always the precursors of gangrene or putrid disintegration.

According to the same author, the shreds of parenchyma which often appear in the sputum contain: (1) Fat crystals, in some parts very numerous, in others altogether wanting, in the form of spherical *Drusen* of about the size of a pulmonary epithelial cell and glistening, refractive appearance; (2) yellowish-brown or brownish-red particles of pigment in varying quantities; (3) crystals of hematoidin, partly in the shape of the well-known rhomboid forms characteristic of bilirubin crystals, partly as delicate bundles of fine needles which radiate in one or two directions and often contain a rhomboid crystal at the center.

Fever is present in most cases of abscess of the lungs; it is mostly irregular; rarely intermittent, and, if so, for a short while only. If the abscess is the product of genuine pneumonia, the latter may terminate by crisis and very soon a fever of varying duration may follow.

The most frequent *complication is pleurisy*, mostly of a purulent character. The complication depends on the seat of the abscess; if it is immediately under the pleura, pleurisy is never absent, provided the two pleural layers are not adherent.

Senator has observed *subcutaneous emphysema* in one case of abscess of the lungs, after pneumonia of the left side. He ascribes it to the passage of air through the external wall of the abscess, which was adherent to the costal pleura, at a point where rupture by means of very fine openings had occurred at "one or several places." The openings allowed air to pass, but only infinitesimal quantities of the fluid contents of the cavity.

As the presence of such minute openings was not positively demonstrated by the autopsy in this case, the question may be asked, Might not the air, in view of the fact that the emphysema appeared first on the left half of the face, have taken its course from the abscess into the mediastinum and spread from there toward the face? In a case of tuberculosis I saw the subcutaneous emphysema which followed hemoptysis appear first under the clavicle, extend from there to the neck and face, and finally spread over the entire body. From this I concluded that a cavity had opened into that part of the mediastinum which is not covered by the pleura, and the air had first entered this cavity. I regretted very much that I could not make the autopsy in this case. The patient was nineteen years of age and died after six days, from an attack of nephritis.

DIAGNOSIS AND PROGNOSIS.

In the diagnosis the presence of a **tuberculous cavity** must first of all be included. The finding of tubercle bacilli forms the best criterion. The expectoration of large quantities of pus at considerable intervals points rather to abscess. Not without value is the knowledge that abscesses occur most frequently in the lower lobes, tuberculous cavities generally in the upper lobes. According to Tuffier, 80% of all abscesses are found in the lower lobes.

The possibility of tuberculosis being superadded to abscess of long duration is proved by the case described on page 751.

In **bronchiectasis** elastic fibers are absent from the sputum, but otherwise the symptoms may be very similar to those of abscess.

Tuffier correctly says that it is very difficult to distinguish between an encapsulated, especially an interlobar pleural exudate and a closed abscess; *i. e.*, one which as yet has no visible communication with the bronchi. To obtain a correct result the causal conditions as well as the entire course of the disease must be carefully studied. [In the former bronchial breathing, from condensation of lung, without râles, and absent fremitus is likely to obtain.—Ed.]

The prognosis depends chiefly on the pathogenesis. It is most favorable in abscesses which originate in genuine pneumonia. Abscesses caused by large foreign bodies, especially beans, becoming wedged fast in a bronchus run a grave and rapidly fatal course.

TREATMENT.

The treatment of abscess of the lungs until quite recently was purely medical, and even now the **medical treatment** should be principally considered in the majority of cases. This applies, above all, to cases in which an abscess has formed in the course of croupous pneumonia. As has been mentioned on page 486, the abscess is always solitary, and its size ranges from 3 to 15 cm. The most appropriate treatment, according to my experience, consists in the **inhalation of carbolic acid.** A 4 to 5% solution, which in the nebulizer will be diluted about one-half by the admixture of steam, should be inhaled three or four times daily. The apprehension that too much carbolic acid may be absorbed is unfounded, as it is prevented by watching for the characteristic dark color of the urine. I have never seen such a change occur even after using the acid for weeks. In addition to this procedure, a nutritious diet and absolute rest in bed are required.

In abscesses due to embolism, bronchiectasis, or the aspiration of foreign bodies, **quinin** may be used, in addition to the inhalation of carbolic acid, to combat the fever which is sometimes accompanied by chills.

Apoland advises in pulmonary abscess to make the patient lie in a **position** that assists the discharge of pus, although at first the cough is thereby aggravated. The patient should bend over every two hours or less, according to the quantity of the discharge. Whether this method has any value I do not venture to decide.

The indications for **surgical interference** can scarcely be summarized, but have to be considered carefully in every individual instance. Whenever the presence of a *secondary empyema* is demonstrated it is advisable to evacuate the pus in every case of pulmonary abscess. In a fatal case, in which I had to give an opinion on the postmortem findings by order of the court, an abscess as large as a plum and an empyema had formed in consequence of a stab wound which had penetrated the lungs below the point of the scapula between two ribs. In this case an operation should have been performed and recovery might have been possible. In the above case of aspiration of an acorn-shell an empyema had also formed, and was evacuated by resection, but the process of disintegration caused by the foreign body remaining fast could not be arrested, and finally caused death after tuberculosis had been superadded.

In addition to resection for the purpose of evacuating a complicating empyema, which is the principal indication for surgical interference, the latter is advisable in cases of *uncomplicated abscess*, if, after its presence has been positively determined, there is no prospect of spontaneous rupture into the air-passages or the general condition and the fever render an operation imperative.

The value of such a procedure is proved by the reports of Runeberg, Quincke, and Tuffier, based on their own observations and on carefully collected statistical material. The last-named author, who has collected the cases from the literature up to 1897, reports the result of operations in 49 cases of pulmonary abscess, 47 cases of bronchiectasis, and 11 cases of pulmonary abscess following the aspiration of foreign bodies.

Of the 49 cases, 23 appeared after fibrinous pneumonia, 6 were caused by septic embolism "in the course of an acute infection"; in

5 cases foreign bodies had been aspirated, and 3 were secondary to suppuration in the neighborhood (liver, kidney).

According to the view of these authors, which is undoubtedly correct, the evacuation of the abscess by puncture should be rejected in favor of **resection** of one or more ribs.

According to Tuffier's compilation, the resection of one rib furnished a large enough field of operation in 19 cases; resection of several ribs was only necessary in 9 cases. In most cases the layers of the pleura were adherent; in 4 cases there were partial adhesions; in 9, the pleuræ were free. Suture of the pleura was done 3 times. In four other cases pneumothorax set in at the moment of operation; in two cases it was only partial and was not followed by grave consequences; but in one case it rendered the operation very difficult, and hastened the death of the debilitated patient, and in another case made it impossible to open the abscess. Five operations had to be performed at two sittings, three of them with the aid of Quincke's method—*i. e.*, the introduction of a paste of chlorid of zinc. In another case tincture of iodin was used to produce adhesions. Neuber resected three ribs, tamponed the wound, and then waited five days before he performed pneumotomy.

Out of 43 operations, 33 recoveries occurred. In seven cases the abscess was not opened; only one of these patients recovered.

The **dangers of the operation** are, according to Tuffier, insufficient diagnosis and the absence of pleural adhesions.

The **prognosis** depends chiefly on the cause of the abscess and the time of operation. Metastatic abscesses offer a worse prognosis than primary ones. The former show a mortality of one-third, the latter of one-fourth of the cases. Furthermore, the timely operation of acute abscess gives better results than the later operation.

Little encouragement is given by the results of surgical interference in **bronchiectasis,** which I include here on account of the indications, which frequently correspond with those of the treatment of abscess. According to Tuffier, out of 47 cases, 13 died; and of the remaining 34, scarcely 7 recovered completely. But it must be considered that in almost all cases pneumotomy and drainage are followed by a material diminution of the expectoration, cessation of the daily evacuation of pus, and, above all, the disappearance of the fetid odor.

At the same time, it is peculiarly difficult to select the right moment for surgical interference in bronchiectasis. Lately I had to treat the laborer K., forty-four years of age, in the city hospital for bronchiectasis in the left lower lobe. He was put on the pension list by the Government Insurance Board on account of cough and dyspnea five years ago. During the first weeks of his stay in the hospital (July 21 to September 1, 1898) he had very high fever, with great weakness; he was very much reduced, his weight was 45 kg. The expectorated sputa were very putrid, but did not contain any tubercle bacilli. Sometimes the patient was somnolent. I was considering a surgical operation, but gave up the idea partly on account of the extreme weakness, and partly on account of the results of percussion over the left lower lobe, which alternated rapidly between dulness and resonance, showing that the accumulated pus was evacuated without difficulty. I recommended only inhalations of carbolic acid and a nutritious diet. The former was carried out three times a day for five weeks, when it was discontinued because the putrid smell had ceased and the discharge had diminished. A week later the inhalations were resumed on account of the reappearance of the odor and kept up for three weeks. When discharged, the patient felt much improved and was out of bed all day. He had been free from fever for seven weeks, although the temperature was quite high during the first weeks of his stay. His weight was 62.5 kg., an increase of 17½ kg.

The results of operations in pulmonary abscesses due to the aspiration of large foreign bodies must be considered very unfavorable, according to Tuffier's statistics. It is true the chances of recovery in such cases are not improved by the omission of any interference. Although in the above case, as in many others reported in the literature, the aspirated foreign body—in this case an acorn-shell—was spontaneously expelled after some time, the process of disintegration in the lungs had meanwhile become so extensive that neither complete recovery nor a long duration of life could be expected.

[A discussion on the surgery of the lungs will be found at the end of the article on Gangrene.—Ed.]

GANGRENE OF THE LUNG.

PATHOGENESIS AND PATHOLOGIC ANATOMY.

Laennec has distinguished two varieties of gangrene of the lungs—*circumscribed* and *non-circumscribed*. In the latter the pulmonary tissue is more moist and more friable than is normal, its consistency is like that of pneumonic tissue in the first stage of edema of the lungs, or in postmortem engorgement with serum. The color shows different tints, varying between a dirty white or light greenish and dark green or almost black. Some parts are softened, disintegrated into a putrid pulp. On section a creamy, turbid, grayish-green fluid is discharged, which smells gangrenous. The affection occupies at least a large part of one lobe, sometimes the greater part of a lung. In some places the healthy or almost healthy tissue of the lungs merges gradually with the gangrenous parts; in other places a zone of primary inflammatory engorgement intervenes; rarely and in some spots the latter has advanced to hepatization. In *circumscribed* or *essential* gangrene only a small part of the lungs is affected. A portion of the pulmonary tissue, irregularly defined and of varying size, is then either in a condition of fresh mortification or one of gangrenous eschar formation, which later becomes colliquative; finally a cavity remains after the gangrenous tissue has become completely softened and cast off.

In gangrenous *eschar formation* the mortified part presents a black appearance with a tinge of green; the tissue is more moist, more compact, harder than that of the lungs, the appearance is exactly like that of an eschar on the skin in consequence of corrosion by caustic potash; the diseased tissue emits the characteristic gangrenous odor. The contiguous portion of the lung to a certain distance shows the inflammatory engorgement in the first or second stage. If the process of decomposition is carried still further, a kind of blackish, greenish, or brownish pulp is produced, which may rupture into an adjacent bronchus. The surrounding inflamed tissue becomes consolidated at the border of the mortified section or forms a pseudomembrane. Occasionally the cavity is crossed by blood-vessels of considerable size, or the latter end abruptly on the walls of the cavity, which may then contain some coagulated blood.

This description is the most correct and thorough, according to my opinion, that has so far been given of gangrene of the lungs. A later description of the causes of gangrenous processes in the lungs by Traube embraces only the circumscribed forms of Laennec, leaving out the non-circumscribed form. At least circumscribed areas only can arise if the gangrene, according to his statement, originates (1) in the vessels, (2) in the bronchi, (3) in already existing cavities created by destruction of the parenchyma.

The origin of gangrene in the *vessels* Traube correctly ascribes to the presence of fibrin plugs containing septic material from other parts of the body; in other words, to embolic processes.

The origin of the affection is explained as follows: In the *bronchi* the sputa stagnate in dilated or non-dilated bronchi and are disintegrated under the influence of high temperature and oxygen. This leads to inflammation of the mucous membrane of the originally affected bronchi, and to fetid bronchitis in the bronchi in which the decomposed masses are deposited; the latter then spreads to the surrounding tissue, which also undergoes septic decomposition.

But here it appears that Traube failed to discriminate sufficiently between putrescence of the bronchial contents and gangrenescence of the pulmonary tissue. Differences between these processes can, however, be demonstrated in many cases, and may also be utilized clinically for the diagnosis between putrid bronchitis and gangrene. This will be further discussed in connection with diagnosis.

But I do not mean to question the mode of origin of the gangrenous process in the bronchi, even in such as are sound. I myself, in the section on Aspiration Pneumonia, have described a case (page 624) in which a gangrenous disintegration set in in the inflamed areas which were caused by aspiration, and probably such a result would be more frequent under these circumstances if the fatal termination were not so rapid in most cases of aspiration pneumonia.

In such cases, as Hensel says, the gangrenous germs themselves must produce a necrosis. "The putrid properties of the aspirated particles lend a gangrenous tendency to the catarrhal pneumonia which they produce; *i. e.*, the contents of the alveoli are destroyed not by a simple necrotic, but by a putrid process. The masses of debris that fill two adjacent alveoli, as they increase, crush the vessels of the intervening wall; the latter decomposes and gangrene attacks the lung itself. The gangrenous process extends in two ways: other places are attacked in the same way as the original focus, and the growing thrombi in the arroded vessels, whose walls have been destroyed, continue to exclude new masses of tissue from nutrition and predispose them to gangrenous disintegration."

Worthy of notice is a remark of Volkmann in emendation of Leyden's statement (page 390), that in rare cases *metastatic gangrene* of the lungs occurs, probably from small thrombosed veins in the petrous bone, in connection with caries of the ear, and that, although it is impossible to demonstrate the existence of a thrombosed vein or of the metastatic abscess, or to discover any other imaginable cause, such a causal connection must be assumed. He says: "I have reason to surmise that in these cases the gangrene of the lungs often originates by the direct passage of the carious ichor from the Eustachian tube into the pharynx and trachea, as sometimes occurs in ichorous carcinoma of the jaw and of the tongue, in pharyngeal diphtheria, or after extensive operations in the ori-nasal cavity. I have seen gangrene follow twice in such cases immediately after a full dose of morphin, given to relieve the patient for one night from the tormenting cough which was kept up by ichor dripping on the glottis."

The necropneumonia which sets in after *perforation of an adjacent organ* must be attributed to an analogous process—namely, aspiration into the air-passages. It originates most frequently in carcinoma of the esophagus; but it is possible that perforation may be brought about by foreign bodies which remain fast in the esophagus, or even by probing the latter.

The third source of gangrene mentioned by Traube—namely, already existing *cavities* in the pulmonary tissue—may, according to his opinion, be due to acute or subacute pneumonia, hemorrhagic infarct, or tuberculosis.

The relations between acute genuine pneumonia and necropneumonia, according to this opinion, presuppose the formation of cavities, while other authorities have assumed a direct transition from pneumonia to gangrene. On the whole, such a transition does not appear to be frequent. Grisolle, in his report of 305 cases of pneumonia, which forms the basis of his essay, found none that was followed by gangrene; and, vice versa, in a series of 70 cases of necropneumonia, which he found described in different works, there were scarcely 5 which, strictly speaking, could be taken to be instances of pneumonia terminating in pulmonary gangrene. In almost all cases gangrene followed after several days' illness in the course of a very mild bronchitis which was often unaccompanied by fever.

Grisolle remarks, in addition, that while gangrene is rarely the consequence of pneumonia, pneumonia is often caused by gangrene. It can, in fact, be observed that the intact pulmonary tissue around the gangrenous area becomes inflamed, and this consecutive inflammation is nearly always the main cause of death, or at least hastens the fatal termination. In the 1501 cases of croupous pneumonia on which I have based my work I have not seen a single case of gangrene.

Traumatism is another cause of gangrene of the lungs. It may be accompanied by perforation of the thoracic wall, or merely by indirect laceration or contusion of the pulmonary tissue. Leyden reports several well-authenticated cases of this nature. Schneider reports a case of gangrene which set in after a bullet wound, and Friedreich one occurring after a contusion by railroad-car bumpers. Both these cases deserve to be specially mentioned because of their terminating in recovery. Necropneumonia may also occur in connection with carcinoma of the lungs. Epstein has exhaustively reported such a case.

It follows from the above discussion that two different kinds of pulmonary gangrene may be distinguished: the **circumscribed** and the **diffuse.** The former may begin in the vessels, when it is of an embolic character; or commence in the finest bronchi, when it is due to aspiration; or directly affect the pulmonary tissue, as in perforations from adjacent organs. The second, the diffuse form, is less known. Is it, as is most probable, due to a catarrhal pneumonia the further peculiar course of which is dependent upon the complication by specific bacteria? May we in these cases ascribe the appearance of the specific causes of the disease to aspiration, while in the circumscribed forms the infection is conveyed by liquid or solid material?

According to my experience, I feel inclined to answer these questions in the affirmative, but do not claim to render a positive decision. I will only mention that my few cases of diffuse gangrene were invariably preceded at varying intervals by bronchial catarrh, which suddenly became aggravated and complicated by the appearance of fever and the expectoration of sputa, the appearance and odor of which presented the characteristics of gangrene, while absolute dulness and crepitant râles could be observed over the lower section of one lung. It is of course impossible to affirm that the specific causes of the disease had entered the lungs by inspiration only, unless the other recognized means of importation—embolism, aspiration, immediate penetration into the pulmonary tissue by perforation—can be excluded.

Judging from two cases that came under my observation, it seems to me that only the excessive, long-continued use of brandy is of importance as a predisposing factor in diffuse gangrene. In both cases chronic bronchial catarrh had been present for some time; one of the patients was a man from the better class of society.

A valuable proof of the fact that pulmonary gangrene may occur as a diffuse affection I consider Mosing's report of the epidemic, or rather endemic, appearance of necropneumonia in the prison of Lemberg in 1842. This report is contained in the Austrian government medical annals, which are very difficult to obtain, and which I only succeeded in securing by the kind efforts of my publisher, Mr. Alfred Hölder. The careful perusal of this report entitles me to agree with the opinion of the author, that the pulmonary affection which occurred in a great number of cases was a "specific inflammation with a marked tendency to gangrene." Although the characteristic signs of gangrene did not develop in all the cases, and "the very peculiar process which precedes gangrene caused the fatal issue or terminated in recovery" (14 died out of 68 affected), there can be no doubt that this inflammation also terminated in gangrene, when one reads that the autopsy of one case, which clinically agreed with all the others, showed that "the left lower lobe was so rotten that, upon taking it out, the finger of the pathologist suddenly penetrated deep into the substance, and a thick ichor, looking like coffee and milk, forced its way out and spread an odor like that of ichor in malignant tumors. The quantity amounted to about two pounds, but no cavity was found, because the entire substance appeared to have been converted into pulp and ichor; the substance of the upper lobe was changed into exactly the same kind of ichor, so that the entire pulmonary tissue could scarcely be recognized." It was also stated that "some of the patients died of a hectic fever with suppuration of the lungs and infected the atmosphere in their neighborhood by their offensive breath and fetid, discolored sputa."

Finally, it may be said that statistics of necropneumonia according to age and sex will be of no value until its different forms, carefully distinguished, can be used as a basis for such a calculation.

ETIOLOGY.

While the conditions named so far for the appearance of circumscribed as well as of diffuse gangrene of the lungs should rather be regarded as predisposing factors, we may look upon the action of special **bacteria** as the direct cause of the disease, according to our present views and experience. Leyden and Jaffe have found several different kinds of **fungi** in the areas of pulmonary gangrene. One of them they conceived to be a descendant of Leptothrix buccalis, and named it Leptothrix pulmonalis; another they described as a kind of fungus consisting of fine, delicate, spiral threads.*

Bonome was probably the first to comply with the requirements of a correct bacteriologic investigation of the cause of pulmonary gangrene. He was able to demonstrate, in eight cases of gangrene of the lungs, Staphylococcus pyogenes albus five times and Staphylococcus pyogenes aureus three times, in pure culture; in one case both kinds appeared together in the same necrotic area. These various **staphylococci** he believes to be the causes of pulmonary gangrene.

Hirschler and Terray found in the sputa from three cases of pulmonary gangrene a kind of *microphyte* which has never been described. It belongs to the class of micrococci and is of almost the same size as Staphylococcus. On gelatin it forms a culture which on about the eighth or tenth day appears similar to four-leaved clover or a flower with six petals. Liquefaction is very slow. On all culture-media a very peculiar penetrating odor, exactly like that of gangrenous sputum, develops quite early and is retained permanently by the cultures. That the development of this fetid odor depends on the presence of putrefied matter produced by the bacteria is proved by the fact that indol and skatol, the products of albumin putrefaction, could be constantly demonstrated in the cultures. This fact fully harmonizes with the experience that in the products of distillation obtained from fresh gangrenous sputa, indol, skatol, phenol, crenol, oxyacids, hydriothon, ammonia, and, among the volatile fatty acids, formic acid, can be demonstrated.

These authors were able to obtain pure cultures of these micrococci from the tissue-juice of the lungs in a fatal case of pulmonary gangrene; and demonstrated abundant quantities of the same in sections of the interalveolar connective tissue by using a double stain. They proved by experiments on animals that this kind of bacterium is pathogenic and causes circumscribed necroses in the pulmonary tissue; but it is not stated that it causes the characteristic odor of gangrene also.

Babès believes that *various kinds of bacteria* may lead to gangrene of the lungs. He was in a position to demonstrate the presence of a streptococcus or a staphylococcus of extraordinary virulence in several cases of pneumonia presenting certain peculiar symptoms. He also found two kinds of bacteria capable of producing pulmonary gangrene: a variety of the bacillus of malignant edema, and a special saprogenic microbe which is similar to Bacterium coli commune, but liquefies gelatin and experimentally causes progressive gangrene. But, as a rule, microbes cultivated from gangrenous foci lose their virulence; one can produce gangrene in animals only by injecting some of the gangrenous mass. He presumes that a combination of different bacteria is necessary for the production of the disease. It also appears that these micro-organisms can be effective only when the lungs have previously been under the influence of other bacteria. No causative relations could be positively demonstrated between diphtheria bacilli and gangrene.

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*See paper by Pearce, on Leptothrix infections.—Ed.

The lack of uniformity in all these findings proves only, according to my opinion, that the bacterial causes of gangrene have not been as yet positively demonstrated.

DIAGNOSIS AND SYMPTOMATOLOGY.

If I give precedence to diagnosis, I do so because the most important symptom of pulmonary gangrene, the peculiar odor of the breath and of the sputa, without which the affection cannot be demonstrated at all, must first be described as exactly as possible, to distinguish it, as well as can be done, from another peculiar odor of the breath and of the sputa which is characteristic of another affection of the lungs that is not gangrenous.

I refer to the differential diagnosis between *putrid bronchitis* and *gangrene,* and in many, possibly in most cases, the difference in the **odor** of the breath and of the sputa is, according to my experience, the most important diagnostic point.

It is true that the determination of this symptom with the aid of the olfactory organ is purely subjective and empiric, but, as it has repeatedly enabled me to arrive at a diagnosis, it deserves to be made use of for want of a better one. The difference between the fetid odor of putrid bronchitis and that of pulmonary gangrene is best described by a simile. In the former, breath and sputa smell like cheese in the different stages of putrefaction; in the latter, like dung-water. Whoever has stood near a gardener while he watered his flowers with this fluid will not deny that it has an unmistakable peculiarity. To characterize the odor in the two diseases I should recommend the adjective "putrid" in putrid bronchitis, and "fetid" in gangrene, designations that have so far been used promiscuously.

It may be confidently expected that in the future better means than are offered by our olfactory organs will be found for the discrimination between the two affections, when it shall be possible to obtain pure cultures of the bacteria which are the cause of putrid bronchitis and to discover their variation from the causes of gangrene. This view is justified by the results obtained so far, which, however, require further confirmation. Lumniczer, while observing all the rules required for a correct bacteriologic investigation, succeeded in cultivating from the sputum expectorated in putrid bronchitis, on agar, small hemispheric colonies rising above the level of the medium and consisting of small bacilli. They were about 1.5 to 2 μ long, slightly curved, rounded at the ends, thickened at the center, and supplied with spores which were quite visible and stained easily with anilin dyes. After a growth on agar of six to seven days the culture developed an odor exactly like that of putrid bronchitis. The author compared this odor with that of acacia blossoms or that of a "rotten" apple blossom. I regret to say that this odor is entirely unknown to me. The pathogenic effect of this bacillus can be proved experimentally. Injected into the pulmonary tissue or introduced into the bronchi through the trachea, in which catarrh had been produced by inhalation of ammonia, it led to a purulent inflammation going on to necrosis.

The **sputum** also presents other essential points for the diagnosis of gangrene. Traube describes the sputum as follows:

The fetid odor of the sputum, the large quantity, the dirty, greenish-yellow color, the tendency, due to its liquefying action on the menstruum, to separate after a time into layers,—an upper, greenish-yellow, opaque, frothy layer; an intermediate, translucent, albuminous layer, of almost serous consistency; and a lower, opaque, yellow layer, exactly like a simple purulent sediment, and consisting of swollen pus-corpuscles and their detritus,—and, finally, the presence in the sputa of dirty, yellowish-white, semi-solid plugs varying in size from that of a millet-seed or flax-seed to that of a bean, with a smooth surface and peculiarly offensive odor, in which microscopic examination reveals the fatty acid needles first seen and described by Virchow in putrefying animal matter,—all these phenomena may occur as well in chronic bronchial catarrh with dilatation of the bronchi as in pulmonary gangrene. The presence of such sputa proves no more than that a process of decomposition (putrefaction?) is going on within the respiratory apparatus. But the question is whether this process takes place within the intact bronchi or is connected with the destruction of the pulmonary parenchyma. When the latter can be positively demonstrated, and then only, we may assume that a process of disintegration analogous to, or identical with putrefaction—*i. e.*, gangrene—is going on in the pulmonary parenchyma. Traube knows but three circumstances from which, under the supposed conditions, a destruction of the parenchyma of the lungs may be inferred: (*a*) If metallic phenomena are present, which —as shown by the form of the thorax, by the percussion note, the position of the adjacent organs, etc.—must be referred to cavities in the pulmonary parenchyma and have developed, so to speak, under the eyes of the observer, *i. e.*, acutely. If, on the other hand, these phenomena are already present when the case comes under observation, they may be due to chronic pneumonia which some time previously has led partly to abscess, and partly to induration of the parenchyma of the lungs with dilatation of the bronchi without the presence of a trace of pulmonary gangrene. (*b*) If the sputum is of the nature just described and contains microscopic bundles of elastic fibers which present the grouping of those which compose the pulmonary parenchyma. It must, however, be mentioned in this connection that elastic fibers occur comparatively rarely in gangrenous sputum, because they appear to dissolve in the gangrenous ichor of the cavities. (*c*) If in the fetid sputum irregular, mostly oblong, gray shreds can be found of punk-like consistency, intersected by fine lines and dots, in which the microscope reveals a large quantity of closely crowded but free molecules of black pigment within an amorphous translucent mass. It seems that the above-mentioned semi-solid plugs containing needles of fatty acid form only when the decomposing animal substance, by defective expectoration or on account of the saccular nature of the cavities, finds an opportunity to remain for some time in the area of disintegration and to become inspissated. Therefore these plugs are absent in pulmonary gangrene, even when the expectoration is abundant and spacious spherical cavities are present.

Fever I never found to be absent in gangrene. It often attains a considerable height—40° C. (104° F.) and over. Often it is remittent, exactly as in genuine croupous pneumonia.

Cough is mostly very tormenting, and gives the patient no rest day or night if the proper remedies are not given. This is due chiefly to the peculiar properties of the secretion originating in the gangrenous area. It is more acrid, more corrosive, than any other secretion caused by the disintegration of pulmonary tissue, and consequently it produces a more intense and more lasting irritation of the nerves of the trachea and of the mucous membrane of the larynx.

The **posture** of the patient is, according to Leyden, inclined toward the affected side. Instinctively a position is tried in which the irritating secretion is not continually discharged, but accumulates gradually and is evacuated at intervals by paroxysms of cough. In addition to the lateral position on the affected side, the patient usually assumes a half-sitting posture if the gangrenous area is in the lower lobe; if it is in the upper lobe, however, the patient is often found in the horizontal position with the shoulders lower than the body.

The *general condition* of health varies according to the duration of the disease and the preceding pathologic conditions; it is in no sense characteristic. Often the patient's appearance does not correspond to the gravity of the disease. The face may be very congested; sometimes cyanosis is present.

In regard to the *strength*, mistakes are likely to occur if it is judged merely by the condition of the patient when he rests quietly in bed. Neither the respiration nor the pulse are accelerated; the voice is strong. But if the patient leaves the bed, to evacuate the bowels, for instance, dyspnea will set in which seems remarkable in comparison to the state of rest; the pulse then becomes very frequent and small; the exhaustion is greater than was to be expected, and it is some time before the former state of rest is restored after the patient returns to bed.

Hemoptysis is a frequent occurrence. It may be comparatively slight, in the form of an admixture of blood in the sputum, or copious, in the form of large quantities of pure blood. The former is a consequence of severe cough, and probably only the result of capillary hemorrhages from the decomposed tissue of the gangrenous area, while the latter is caused by the extravasation of blood from large eroded vessels which are not completely thrombosed.

Percussion usually enables one to demonstrate extensive consolidation, both when the gangrenous process is diffuse, and when the focus is originally small, because in the latter case extensive pneumonic consolidation of the surrounding tissue very soon develops. The dulness rarely corresponds to the area of the individual pulmonary lobes. Large gangrenous foci often occupy the lower section of the upper, and at the same time the upper part of the lower lobe; or the lower part of the middle, and a part of the lower lobe. Neither is the intensity of the dulness uniform over the affected section. A tympanitic element may be added, and serve, in addition to the contents of the sputa, to indicate more extensive disintegration of tissue.

Auscultation usually shows the presence of crepitant râles in addition to changes of the respiratory murmur going on to bronchial breathing.

The most frequent **complication** is *purulent pleurisy* which may result in copious purulent or ichorous exudates. Its appearance depends mostly upon the original seat of the gangrenous area. The nearer to the pleura this is, the more likely is an exudate to complicate the condition.

Among other complications the occurrence of *embolism* in the peripheral organs (brain, kidneys, stomach) should be mentioned. The emboli originate in thrombosed veins in the neighborhood of the gangrenous area.

All the above symptoms occur both in diffuse and in circumscribed gangrene, and are therefore of no value for the distinction between the two forms. Careful inquiry into the history and the etiologic conditions which have been exhaustively discussed is needed to enable one to make a diagnosis.

If after puerperal fever or a surgical operation a previously healthy lung becomes diseased and fetid sputa make their appearance, the occurrence of gangrene due to embolism should be suspected. Gangrenous foci may originate by aspiration of the contents of the mouth through the bronchi, as in the case described on page 624 in the section on Aspiration Pneumonia. Perforation of the esophagus may lead directly to the formation of a gangrenous area in the pulmonary tissue; gangrene may also develop directly from an already existing focal disease of the lungs, especially if tuberculous cavities are present.

Diffuse gangrene, according to my experience, is generally preceded by bronchial catarrh of varying duration, while all causal conditions for a focal gangrene are absent. The catarrh is at first afebrile; later, fever sets in, either gradually or with a chill, and after dulness has been discovered, usually over a lower lobe, the fetid sputa make their appearance. The presence of a high degree of alcoholism is not without value for the diagnosis. [Latent diffuse gangrene with rupture and fetid pleurisy has recently been discussed by Anders and McFarland, with report of a case. The authors believe that the absence of the characteristic symptoms of gangrene was due to the fact that the diseased parts did not communicate with a bronchus.—Ed.]

TREATMENT.

While formerly the prognosis of gangrene of the lung was considered to be very unfavorable, and indecision accordingly prevailed as to the proper therapeutic measures, since Skoda's report of the success in the treatment of this disease quite a number of cases of cure, or at least comparative amelioration, can be recorded. In addition to internal treatment, favorable results have recently been attained by surgical interference.

Inhalations of turpentine, first employed by Skoda, have been replaced by **carbolic acid.** The latter is recommended by Leyden instead of **creosote** solution which was formerly used. I have used 4% and 5% solutions according to his suggestions. The atomizer reduces the strength by one-half. I have never used the remedy internally. Leyden prescribes solutions of ¼% to 1%, with *peppermint water* or *oil* to disguise the taste, to be taken in tablespoonful doses internally. I prefer Traube's suggestion, to give **lead acetate,** 3 to 5 cg. (½ to 1 grain) with 5 dcg. (7 grains) of *sugar* every two hours or three or four times a day; it appears to be especially valuable when the sputum contains blood. Intercurrently **quinin** may be used, because, according to Binz, its effects are antipyretic as well as disinfectant in septic processes. Leyden gives it every two hours three or four times in succession, ½ gm. (7 grains) at a dose.

For the *cough* **morphin** is indispensable, because otherwise the patients become exhausted by the broken rest at night and by the exertion of coughing. I cannot agree with the view that the use of opiates causes the ichorous secretions to accumulate and thus become injurious; I rather think that opiates, especially morphin, in addition to allaying the nervous sensibility, tend to restrict the quantity of secretion. It goes without saying that in every instance a **nutritious diet** and, if necessary, **stimulants** must be given.

Surgical interference is indicated in pulmonary gangrene, especially when it is complicated by *empyema.* According to my experience,—which, however, consists of a single observation,—and according to the reports of others, I am inclined to consider this complication a favorable result; at least, out of 8 cases cited by Tuffier 7 recovered. In my case the circumstances were practically as unfavorable as in severe cases of gangrene without empyema; yet the patient made a satisfactory recovery. I therefore give a full report of my observation:

Emma Kl., twenty-three years of age, born in Rostock, has always been healthy except for an attack of measles which she had when five years old. On November 27, 1882, she was delivered of a mature child without artificial help. On the third day of the childbed severe chills set in, followed by fever, but there were no pains in the abdomen. On the fifth day the chills recurred, and she was attacked by stitch in the side and severe cough. Fourteen days after confinement she was admitted to the hospital.

Condition on December 10, 1882: The patient looks very pale, extremely emaciated, and creates the impression of great prostration. Her sensorium is a little clouded. She complains of stitch in the left side, which is not severe, however. The abdomen is soft, not painful even on strong pressure. On vaginal examination the presence of an iodoform tampon can be demonstrated, otherwise only a moderate discharge which is not fetid is present. The heart is without abnormality; the lungs are also normal, except the left lower lobe, over which slight dulness, increased pectoral fremitus, bronchial respiration, and tympanitic râles are present. In consequence of the severe cough, large quantities of pneumonic-looking sputa are expectorated. The temperature is very high. On December 12th it rises to 41.2° C. (106.2° F.). The urine is free from albumin and remains so during the entire duration of the disease.

In spite of the use of quinin the fever remains very high during the following days. The thermometer shows almost continually 40° C. (104° F.) in the evening. Continuous diarrhea and great prostration prevail until December 23d, and on this day commencing decubitus can be observed.

On December 27th dulness and diminished pectoral fremitus are present posteriorly on the left side, from the spine of the scapula downward. The heart is displaced to the right. Twenty-four hours later a great quantity of thick, putrid fluid is suddenly expectorated. Immediate examination proves that the dulness behind on the left side now only reaches to the middle of the scapula. On puncture with the Pravaz syringe a dirty-gray liquid which has the same smell as the expectoration is obtained.

On December 29th thoracentesis is performed in the sixth intercostal space and in the posterior axillary line and 1220 c.c. of a thick, purulent, turbid fluid of the same smell are evacuated in the course of two hours. On the following day, December 30th, the patient has no fever whatever, the temperature in the morning was 36.2° C. (97.2° F.), in the evening 37.6 C°. (100° F.); but it soon rises again, and on January 3d the former degree of fever is reached. The dulness again reaches to the spine of the scapula.

On January 6th the thoracic cavity is widely opened by costotomy under chloroform narcosis and a very abundant quantity of the above-described fluid evacuated. After washing the thoracic cavity a drainage-tube is introduced and the wound dressed. The temperature goes down from 39.6° C. (103.4° F.) to 36.2° C. (97.2° F.) in the evening, and, as I may state here in advance, after staying at about 38° C. (100.6° F.) for ten days it remains normal from then on.

January 7th, irrigation with thymol causes collapse and severe nausea. Supposing that the thymol was the cause of the collapse, on the following day a solution of salicylic acid (1 : 300) is used for irrigation. Then the patient states that the water reaches her mouth. Consequently, although at that time irrigation of the thoracic cavity was considered obligatory, the procedure was omitted, and only a week later a solution of silver nitrate (1 : 10,000) was used as a disinfectant. The patient states, unasked, that she has a metallic taste on her tongue, and therefore irrigation is definitely discarded. Four weeks later the secretion has ceased almost entirely; what remains consists of thick, laudable pus. The percussion note posteriorly has become clear except over a zone three fingers wide in the lower portion of the lung. Only slight cough prevails. On February 18th a 1 % ointment of silver nitrate is injected. Two days later the incision wound has closed permanently. The cough continues with thick, greenish, inoffensive sputum; it is allayed within a few days by morphin powders.

On March 11th the patient leaves the hospital. She looks very well and has gained considerably in weight, 4.5 kg., from February 6th to February 27th. Posteriorly on the left side, from the spine of the scapula down, the percussion note is somewhat shortened, and weakened vesicular breathing is present; a little above the scar the note is slightly tympanitic, the respiration slightly bronchial; catarrhal sounds are nowhere to be heard.

Six months later the patient once more presents herself. She is perfectly well. Nothing abnormal can be demonstrated in the lungs. Two years later she writes me that she enjoys permanent good health.

This case deserves special notice for several reasons. Its origin is easily explained. The pulmonary gangrene was undoubtedly caused by an embolus which originated in one of the peri-uterine veins, and its entrance into a pulmonary vessel was marked by sudden stitch in the side and cough. But the thrombosis in these veins was connected with the puerperal fever. When the patient was admitted to the hospital, she had pneumonia, as was shown by the characteristic sputa, the dulness, the bronchial breathing, and the increased pectoral fremitus. This pneumonia may serve as an example of the correctness of the opinion, first expressed by Grisolle, that a pneumonia may appear in the neighborhood of a circumscribed gangrenous focus. Empyema then developed, and not until then, four weeks after the embolism, the gangrene manifested itself by the characteristic sputum. After the empyema had been evacuated by surgical means, irrigation of the thoracic cavity proved that the gangrenous area had perforated into the pleural cavity; the irrigating fluid penetrated through the gangrenous cavity into the bronchi, and from there into the mouth. The disease lasted about four months.

In cases of uncomplicated pulmonary gangrene it is difficult to lay down the *indication for surgical interference*, and especially the proper time for this procedure. Tuffier says: "The continuance or the repetition of septic accidents, which must be ascribed to insufficient evacuation of a gangrenous focus,—accidents which affect the general condition of the patient,—have been the cause of surgical interference. The indications vary according to the seat of the focus, the severity of the symptoms, and the cause and progress of the dis-case. In gangrene of the pulmonary apex the danger is very great, and early interference is demanded. Gangrene following the entrance of food-stuffs or partial rupture of the esophagus is generally so rapidly fatal that the operation has always been performed too late. On the other hand, cases of diffuse gangrene, the presence of foci, or of several foci in both lungs, and symptoms of meningitis or of brain abscess are counterindications to operation. The absence of pleural adhesions makes the prognosis more unfavorable, but must not deter us from operation. The most important point is to perform the operation at the earliest possible moment, because this will be the best guarantee for its success."

Accordingly, it may well be maintained that at present operative interference, and especially the proper time for operation, are to a very considerable degree matters of individual opinion. I believe that for the present, in view of the advice to operate early, the *indication* may be concisely stated as follows: Operative intervention is, above all, advisable when persistent high fever, especially if accompanied by chills, has led to a considerable degree of debility, any further aggravation of which, according to general experience, is bound to endanger life; and when the gangrenous focus is so situated near the pleura that there is good reason to expect that it will be easy of access. In stating the indications it must be kept in view that the operation itself may be followed by accidents which are of the greatest danger to life. In every individual case, therefore, the question whether its course might not be more favorable without operation is to be carefully weighed.

The dangers referred to include hemorrhage, which may appear in the course of, or after, the operation. It occurred 9 times in the 74 operated cases on which Tuffier bases his statistics, and caused death 4 times.

In the case described on page 629 in the discussion on aspiration pneumonia I succeeded in arresting a severe hemorrhage by packing the pleural cavity with a strip of iodoform gauze large enough to fill a volume of about 500 c.c., and thus brought about recovery.

On the other hand, the danger due to the absence of pleural adhesions does not appear to be as great as is generally supposed. Among the 74 cases collected by Tuffier the pleural cavity was completely obliterated 54 times; in 14 cases the pleura was not adherent at the site of operation, although adhesions were present in the neighborhood; and in 6 cases they were altogether absent. Two of these 6 cases terminated fatally; but death was in a large measure due to the severity of the disease itself. In the third and fourth cases the gangrenous cavity was opened, in the fifth and sixth cases it was impossible to do so. In one of the two latter cases spontaneous rupture of the gangrenous focus into the pleural cavity took place after two days; in the other, pneumothorax developed. In spite of these accidents, however, both cases ended in recovery.

In view of these results, and in view of the favorable termination, as stated above, in cases in which an empyema is present from the beginning in addition to the gangrenous foci, the opening of the pleural cavity need not be regarded as a particularly unfavorable complication. At least it does not seem to me advisable to waste much time in looking for the gangrenous focus when the pleural cavity has been opened.

I am led to this opinion not only by the favorable results in cases in which the pleural cavity has been opened, and by the frequent recoveries in cases complicated by empyema, but also by the great necessity of abridging the period of anesthesia as much as possible in cases of gangrene that have already greatly exhausted the patient. In a case of gangrene of the left lower lobe,—which, it is true, had already lasted eight weeks, and had greatly weakened the patient by reason of the high fever, the violent cough, and the bloody expectoration,—I opened a large gangrenous focus without loss of time and without difficulty; but, although the anesthesia lasted at most ten minutes, the patient, when he awoke from his narcotic sleep, was completely cyanotic, with extremely small, frequent pulse. The difference in the general condition before and after the operation was striking, and, in the absence of any other cause, could only be attributed to the effect of the chloroform. Although the fever and the cough subsided and the bloody expectoration ceased, the patient failed to rally. Death ensued without any other complication five days after the operation.

The question as to the proper method of procedure in individual cases cannot be regarded as settled.

[NOTE ON THE SURGICAL TREATMENT OF SOME PULMONARY AFFECTIONS.

Surgeons confine themselves to the treatment of certain diseases of the pleura and to the treatment of abscess, gangrene, bronchiectasis, and hydatid disease of the lung, and in rare instances to the removal of tumors (see the treatment of foreign bodies) and the drainage of tubercular cavities. The accompanying bibliography shows their activity in this direction. In addition to Tuffier, whom the author quotes, we have reliable information from the experience of Godlee, recorded in the work of Fowler and Godlee on diseases of the lungs. Eisendrath's is the most recent of American papers. But operative procedures were thought of as early as 1664. Eisendrath points out that two periods belong to pulmonary surgery. The first ended thirty years ago. The second period began in 1873 with Mosler's attempt to cure abscess. The surgeons subsequently were fortified by Schmidt and Biondi's experiments on animals, which proved that pneumectomies were well borne.

In the first period belonged Willis (1664) and Baglivi (1696), the former suggesting the possibility of, the latter recording, a lung operation.

Purman (1692) says chest and lung incisions are necessary in hemothorax, chest wounds, cavities of lung, abscesses, etc.

Bligny (1670) reports a case of phthisis which recovered after an accidental sabre cut.

Barry (1726 and 1763) reports several cases in which incision cured the patient.

Sharpe (1769) says severance of adhesions is not necessary; the cavity should be simply punctured with a lancet and drained.

Ponteau (1783) and David (1783) recommend courageous opening of abscesses.

Operation in Cases of Abscess.—Faye (1797) cured an abscess by operation; recovery very slow.

Richerand (1812) and Jaymes (1813) report cured cases.

Bell (1805) reports a cured case and several failures.

Callisen (1815), Zang (1818), and Nasse (1824 and 1844) advise it; the latter reports successful cases.

Krimer, Bricheton, and Borchert report favorable and unfavorable cases; the former applied caustic remedies to the pleura.

Breschet (1831), MacLeod (1836), Claessens (1839), Hastings and Stork (1844), Herff (1844), and Collins (1855) operated, evidently for empyema.

Mosler (1874) and Pepper (1874) advocated surgical procedures, the latter in tuberculosis.

Bull (1883): Large bronchiectatic cavity of right base; incision in ninth intercostal space; opened lung with cautery and finger; pus escapes through a fistula; death. Autopsy: Numerous bronchiectatic cavities.

Smith (1883) operated for gangrene.

Biss (1884) large right-sided bronchiectatic cavity; death after four weeks. Autopsy: numerous bronchiectatic cavities; largest opened; 2 brain abscesses.

Finny (1884): Gangrene; opened too late; death.

Runneberg: Gangrene; operation; no cavity found; death. Numerous small cavities suggestive of one large cavity.

To obtain good results from surgical procedures a clear conception of **general indications** for and against operations must be obtained. This has been insisted upon by all surgeons. Borchert, for instance, states that the exact situation and limits of the lesion must be accurately fixed. No other foci should exist in the same or in the other lung.

In acute or chronic lung cavities, he further states, whether following upon acute or chronic inflammatory processes, which show no tendency to heal when medically treated and are associated with suppuration, necrosis, and putrefaction, an operation should be resorted to. When there is danger of septicemia, pyemia, or hemorrhage, and when the patient's strength is becoming undermined by constant suppuration.

Tuffier says a precise diagnosis and an early intervention are the two elements for success. Precise diagnosis refers to the seat of the lesion and its limits. Percussion, auscultation, radiography, and exploratory puncture are at our service to assist us. Regarding results, he thinks adhesion of the pleura is very important, and, if not already existing (it does in 87%), should be brought about by sutures; or, if no hurry exists, by the application of acupuncture, caustics, or tampons of iodoform. He prefers the use of sutures.

In general, we have three **methods** of operating on the lung. König's classification includes all pulmonary but no pleural operations.

1. Subcutaneous operation on diseased lungs.
2. Second method, which opens and drains lung cavities.
3. Third method, known as resection of lung.

The percutaneous method of Guérin (1830) was revived by Vidal in 1882: With a glowing iron or a Paquelin cautery he touched the skin over the cavity in a number of places, producing a redness of skin lasting for hours, not limited to the point of application, with relatively little pain, easily recognizable influence on symptoms, etc.

W. Koch, Hiller, Mosler, and Pepper in the early seventies injected subcutaneously into the lung substance medicinal fluids with the intention of improving the local disease: potassium iodid and iodin; silver nitrate; carbolic acid; bichlorid of mercury; salicylic acid; potassium permanganate. Mosler cured an echinococcus cyst with carbolic acid; Maragliano, an abscess of the lung of three years' standing with silver nitrate.

The second method, an old one, was modified by Mosler and Hüter (1873), who were the first to deliberately open a bronchiectatic cavity in the right upper lobe, through the third intercostal space; a drainage-tube was introduced, through which the pus was carried off, and the cavity was then irrigated with carbolic acid solution.

The third method, resection, was made possible by animal experimentation. Glück, Schmidt, Block, and Biondi demonstrated that portions of lungs of animals may be removed and the animals live. Couvy, in traumatic hernia of the lung, ligated the protruding lung, resecting it on the third day: cured after twenty-eight days.

Borchert stated very clearly the *method of operating.* It should be composed of two parts: (*a*) Thoracotomy and pleurotomy; (*b*) pneumotomy. If empyema is present, this should be drained first. If adhesions exist, both stages can be performed at once. If no adhesions are present, they must be produced either by suturing the pleura or by applying chlorid of zinc paste after Quincke. He prefers the former. Thoracotomy should be large—of at least the same size as the disease of the lung. Healing occurs by granulation. To facilitate expectoration, the fistula must at times be plugged. The cavity should not be washed out, if it communicates with a bronchus. The wound must be kept open as long as possible by means of drains and tampons.

Eisendrath speaks as follows: The most important point in technic is to decide whether adhesions are present or not; this cannot be positively determined, but in 90% they are present. Quincke even produces adhesions by zinc chlorid paste, but this requires several weeks. Parietal and visceral pleura are or may be sutured during thoracotomy and pneumotomy. Pneumothorax is to be avoided. Several ribs may be resected. Fatal hemorrhage has resulted from the use of the exploring needle. A thickened pleura may interfere with healing and may be removed. Pus may not immediately appear. The cavity may be either tamponed with gauze, or a rubber tube may be used to prevent pressure on the vessels. A secondary empyema simplifies the process. Irrigation should not be employed, as it may carry pus to other parts of the lung. If a large bronchus communicates with the cavity and a fistulous tract remains, open the wound, cauterize the bronchus, and remove that portion of the lung forming the abscess cavity; this has been done successfully in many cases. If the cavity shows no tendency to heal, resection of a number of ribs and compression of that side of the thorax have aided in closing the same; if the fistula remains open, the patient can wear a bottle containing an antiseptic solution.

The methods of operation in gangrene or abscess are formulated by Godlee: After localizing the abscess, an aspirating needle should be introduced again and again, until pus is discovered. There is danger of producing septic pleurisy. Chloroform is used as an anesthetic, after discovering pus. Ether causes an increased bronchial secretion, which, added to the already existing discharge, may produce dangerous symptoms. The needle enables the operator to find the pus; therefore it should be left in. Thoracotomy. Suture of pleura, if no adhesions, until air-tight. Pleurotomy. Pneumotomy with knife, forceps, or cautery. He likes forceps best. Antiseptic packing, drainage, and dressing. Frequent washing of tube; no washing of cavity, however.

Tumors.—Operative measures have not been successful, partly because of the difficulties of diagnosis and also for anatomic reasons. Tuffier's operations were for growths secondary to those of the chest-wall. His results, he feels, are encouraging, as four out of seven patients recovered. The tumor and neighboring pulmonary tissue were removed.

Minlichner's case illustrated the dangers, as the pleura became infected and death resulted twenty-four hours after operation. The growth was a myxochondroma of the right thorax, attached to the lung over an area the size of a small plate. The tumor was tied off and two nodules excised from the upper lobe of the lung.

The next operator was more fortunate. Krönlein removed at a second operation a portion of a lung for sarcoma. The lung was sutured and in three weeks healing was complete. The patient was alive four years after the operation. Krönlein says that if septic infection is prevented the chances for recovery are good.

Reclus does not look upon the removal of tumors with favor, for an accessible, single, circumscribed tumor is a great rarity; no cases are reported, to his knowledge. Krönlein's case, he states, is exceptional, and was secondary to sarcoma of the chest-wall.

Péan, on the other hand, says that if superficial solid tumors of the lung are found they may be removed. He, as well as Krönlein, and also König, have removed portions of the chest-wall and metastases within reach.

Borchert states the truth as follows: No tumors have been diagnosed early enough to permit a successful operation. Successful operations for tumors must therefore be the exception, and, save in rare but fortunate cases, cannot be advised.

Foreign Bodies.—Surgical measures to relieve this condition have been spoken of in the section devoted to foreign bodies in the lungs. Reclus remarks that if the foreign body becomes encysted, no operation is required; but if suppuration sets in, it must be treated by surgical measures. Quincke states that foreign bodies should be removed as quickly as possible; the attempt is imperative if suppuration sets in. Of 7 cases, 2 were cured, 2 improved, and 3 died.

Freyhan's statistics indicate that an operation must be resorted to early. In Hoffman's collection of cases, which consists of 147, 46 were cured by coughing up the foreign body; 7 by the foreign body rupturing externally; 26 through tracheotomy and extraction; 11 died in spite of tracheotomy; 1 died in spite of pneumotomy; 52 were unoperated; 1 improved unoperated; 3 uncertain unoperated.

If left to themselves, abscesses due to foreign bodies will invariably terminate fatally. Freyhan collected 9 operated cases; 4 died, 4 improved, 1 cured. He believes that if operation were performed early, the results would be better.

On the other hand, Fowler's * case of aspirated tooth is classic and encouraging: Formation of abscess in base of right lung; pneumotomy, recovery. Moorhof †: Abscess of left lung following a gunshot wound, through which portions of ribs were carried into lung. Abscess opened five months after injury; death due to metastatic abscesses.

Echinococcus.—We have the authority of Reclus, Thomas, Péan, Doyen-Garré, Fenger, Freyhan, Rüdinger, and Borchert in favor of operative measures. The latter author states that echinococcus should always be operated on, and Rudinger states that operative results are excellent. Mosler and Piper (1894) report twenty-one unoperated cases with 50% of recoveries, but they add the records of operative cases with 90% of recoveries. Freyhan shows that the prognosis is very bad without operation, which Taufort corroborates in his paper.

Reclus states that in hydatid cysts pneumotomy is efficacious and the operation of choice. If not operated on, patients may recover, but statistics are against non-interference.

Neisser reports 61 untreated cysts with 15 recoveries and 36 deaths. Davaine and Hearn believe these unfavorable statistics to be due to expectant treatment.

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* *Tr. Med. and Surg. Soc. Lond.*, May 31, 1874.

† *Wien. med. Presse*, 1889.

John D. Thomas reports 5 personal and 27 collected cases, which were operated on: 27 recoveries, 5 deaths. Lopez, of Lisbon, 36 operations with 31 recoveries and 5 deaths.

Of simple puncture, Maydl reports in 16 cases 5 recoveries and 11 deaths. Deaths were due to purulent pleurisy, pneumothorax, and asphyxia due to rupture of cyst into bronchus. According to Thomas, abstention gave a mortality of 54%, puncture 27%, pneumotomy 16%. He states that nearly all competent surgeons declare for pneumotomy, and reports 11 interventions for hydatid cyst with 9 recoveries and 2 deaths. In several cases broncho-pulmonary fistulæ persisted for a long time. One death was due to asphyxia.

Péan: Primary and secondary hydatids should be opened. Doyen reports 2 cures; Jonnesco, 1 cure.

Rose and Carless *: Incision and drainage have so considerably reduced the mortality that this method alone should be adopted.

Regarding the plan to be pursued, Garré and Sultan † speak forcibly. Pneumotomy is the only operation.

Tuffier and Garré report 79 cases: 71 cures, 8 deaths.

Israel formulated the mandate that puncture of a hydatid cyst is not permissible, as through it such irritation may be produced as to rupture the cyst into the pleural cavity. Pneumotomy is not as dangerous. He refers to one case in which the patient nearly died as a result of puncture; recovery followed, however, after incision and drainage.

Fenger and Hollister's case illustrates the easiest method the surgeon may be called upon to use. It was a gangrenous abscess of the middle lobe of the right lung, produced by suppuration of a large echinococcus cyst. Opened; drained; sac removed; washed antiseptically; recovery.

Jones advises the operation in two stages, as is done in liver hydatids. He punctures only under narcosis, as without it a coughing spell may rupture the sac prematurely.

Bronchiectasis.—Rüdinger well remarks that the difficulties of diagnosis are great and the chances of success few, even if the diagnosis is correct, on account of multiplicity of cavities. No one individual experience is sufficient to form the basis of an opinion; hence a brief of the surgeons who have had experience is herewith submitted. As long ago as 1883 Bull operated, although the patient died. In 1891 Kercke submitted that operation was permissible if only one cavity was present. Usually foci are multiple, however. In this case pus was found by puncture, but at the operation the cavity was not found. At autopsy numerous cavities were found. A pneumothorax occurred during operation.

Taufert ‡ in the same year stated that the prognosis is bad, first, because we never know whether one or more cavities are present; second, because on account of the thickness and rigidity of their walls and their position in the midst of thickened lung tissue, the collapse of the cavity is prevented.

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* "Surgery," 1901.

† *Deut. Arch. f. klin. Chir.*, 1902, XXXII.

‡ "Ueber Lungenchirurgie."

Prior to this it was said by Rochett * that if the bronchiectatic cavity is single and large, and before the sputum becomes gangrenous, operation is indicated.

Reclus stated that pneumotomy had not given good results. The cavities which have been opened improve; but there are usually so many others, which have not been opened. For a time only does the patient improve. Of 5 collected cases, 3 recovered, 2 improved. Indication for operation: (1) If the pulmonary excavation is the essential lesion; (2) if the symptoms of putrid absorption dominate the scene, producing the fever; (3) if cough weakens patient on account of profuse expectoration. We must not promise too much, and consider operation only palliative.

Walther reports operation in 1 case: numerous cavities. Opening of largest one: improvement.

Quincke discussed the subject very exhaustively.

1. Results of operation are less favorable than in acute cases; still, improvement results quite often.

2. Fistulæ are very persistent, and are explained by the long existence of abscesses: the indurated tissue cannot collapse.

3. Abscesses and bronchiectases should be operated on before they become chronic.

4. Extensive resection with possible destruction with caustics gives best results.

5. Adhesions are less frequent; he advises formation of adhesions, although not absolutely necessary: 8 cases—1 cure; 5 improvements, 3 with fistulæ; 2 deaths.

6. Operation should be performed in chronic abscesses and saccular dilatations; the prognosis is less good, but if we do not operate, the condition is bound to get worse. If multiple cavities are present, little can be expected, but they offer some chances, and will get worse if not operated on.

Freyhan collected 25 cases with 13 deaths, 7 no change, 3 improved, 2 cured. When the disease is unilateral, the prognosis is better than when a bilateral lesion exists. He concludes they had better not be operated on, unless unilateral type can be diagnosed.

More recent writers hold to similar views. Garré operated upon 35 cases; 17 cured. Lenhartz operated upon 4 cases; 4 cured.

Borchert says that if bronchiectatic cavities are in a state of ulceration and gangrene, if the size of the cavity, the long time of the suppuration, and the large amount of pus cause debility and threaten amyloid degeneration, the cavities should be operated upon.

Carless and Rose state that if a single dilatation exists with a good deal of fetid secretion, expelled with difficulty, operation may

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* *Wien. med. Presse*, 1886, 1266.

be useful. If many cavities are present, but little benefit can be expected.

Garré reports 5 cases: 3 died within a short time, 1 died within a few months. Discouraging, were it not for the knowledge that with the disease they cannot live long. He thinks pneumotomy should not be performed. Of 59 collected operations, 21 died. Quincke's operation—removal of the overlying chest-wall without pneumotomy—would probably be the best procedure. It gives the lung a chance to collapse.

[SURGICAL TREATMENT OF ABSCESS OF THE LUNG.

Pulmonary Abscess.—We cannot allow this affection to progress, if sure of the diagnosis, without surgical interference. This is positive if the abscess is single, if it is at the base of the lung, and if the drainage by the bronchial tubes is poor. Moreover, all surgeons agree the operation should be performed as soon as the diagnosis is made.

Rose and Carless believe abscesses following pneumonia should always be operated upon. Eisendrath advises in acute abscess to wait four to six weeks, in the hope that nature may bring relief. Freyhan insists on early operation because then the prognosis is very good; it is, on the other hand, very bad in chronic cases. Borchert believes an operation is justified with or without empyema. He thinks the surgeon should not wait too long, but if acute suppuration prevails, with high fever, a tendency to hemorrhage, the occurrence of septicemia or pyemia, and the onset of exhaustion, an operation should be undertaken at once. Rüdinger* operates as soon as diagnosed, whether the abscess is superficial or deep. The operation may be done in two sittings if pleural adhesions are not present.

Garré advocates early operation. Reclus states that while small abscesses may become absorbed, and large abscesses without diverticula and with free opening into a bronchus may cicatrize, cavities large in size, irregular in outline, situated in the base of the lung, with poor egress for pus, must be operated on. Deep situation of the abscess and absence of adhesions must not deter. The indications are the above-described abscesses; the contraindications for operation arise from the small size of the abscesses, their lack of tendency to cicatrize, and the presence of free drainage. He operated 23 times with 13 recoveries and 3 deaths. Reclus points out that men recover easier than women. Twenty of twenty-three of his cases recovered.

Garré collected 96 cases of operated abscess with a cure of 77 and a mortality of 19. That the results are encouraging one can gather from the statistics of Eisendrath. Of the large number of cases collected by him, about 85% recovered from the operation. When one considers how prolonged and how fraught with dangers of all kinds an illness from pulmonary abscess is, and how little risk apparently attends operation, one can readily subscribe to the proposition to operate on all cases at once, if there are alarming or even moderately severe symptoms, or to delay operation only in cases in which we recognize free drainage from the abscess. One need not, apparently, have the dread of pneumonia, as in operations for removal of foreign bodies. We do not read of this occurrence in abscess; we are taught to dread it in foreign-body operations. The danger is less, moreover, in those cases in which the abscess is the result of pneumonia. Thus, of 17% of the cases of Reclus, 14 recovered. In Eisendrath's collection the abscesses due to the pneumococcus infection were in great excess, and the mortality lower than all others.

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* *Deut. Arch. f. klin. Med.*, 1887, xli.

A review of the literature shows that surgeons have considered it essential to weigh the following circumstances:

1. Naturally, (*a*) the diagnosis of abscess, including its distinction from tuberculosis, gangrene, bronchiectasis, and empyema; (*b*) its cause, as influencing treatment and prognosis; (*c*) its accompaniments, whether single or multiple. The reader is referred to the text for a consideration of these questions.

2. The indications and contraindications for operation. The presence of a single abscess is of itself an indication for operation, if it occurs in an operative subject. No other indications need be discussed. However, it is just to remember that the chance for recovery in a pulmonary abscess, if not favorably situated, is better if the operation is delayed until the question of free drainage is decided. In an abscess at the base that has been reached by a needle, the patient is safer in the hands of the surgeon than if left to nature.

3. If the operation is indicated, shall we wait for adhesions to form, or form them artificially? It seems that such delay is not necessary with our present operative technique. Notwithstanding the authority of Quincke, we would prefer to follow the advice of Rüdinger, and operate at once.

4. The chronicity of the abscess increases the danger and lessens the liability to recover. The experience of Hearn and Roe is encouraging, however, for they succeeded in curing an abscess of two years' duration which eluded detection in three attempts to find it.—Ed.]

[SURGICAL TREATMENT OF GANGRENE OF THE LUNG.

Gangrene of the Lung.—As with abscess, so with gangrene, practically the same considerations obtain. Given a single focus, in an operative region and in an operative subject, the diagnosis is sufficient warrant for an operation. Indeed, we should go further in this statement, and in a proper subject hunt for further trouble if there were indications of at least two foci. As Rochester points out, delays are even attended by greater dangers than in abscess. The famous case of Osler and Cans shows that one need not be deterred from excising large areas of sloughing tissue.—Ed.]

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DISEASES OF THE PLEURA.

BY

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DISEASES OF THE PLEURA.

A. ACUTE AND CHRONIC INFLAMMATION; TUMORS.

I. INTRODUCTION.

The development of the doctrine of pleurisy reflects a true picture of the history of clinical medicine and the theories which dominated it; nowhere else do we see so clearly the influence of the theoretic and the empirical, the localizing and the generalizing, schools.

While some regard only the general clinical picture and view the local phenomena merely as the accidental expression of a change in the constitution and in the interchange of body-juices, others seek the true nature of the disease in the local process, and accordingly attempt to determine the local signs of the disease and by them to explain the processes going on in the tissues. While in the past one school considered the tissue changes in the pleura equal in kind and degree to the changes in the pulmonary tissue itself, and accordingly were led to exaggerate the importance of the general symptoms at the expense of those of the local affection; the other school went to the opposite extreme, and, in attempting to account for all the signs of an altered reaction, created an endless number of different forms of the disease—new species, as it were, of an artificial system.

When clinical medicine was dominated by the teachings of pathologic anatomy, this attempt reached its highest development. In the effort to make the results of clinical examination, which at best present a monotonous uniformity, harmonize with the rich variety of anatomic pictures, the widest limits of possibility were reached and an attempt was made to resolve the few main classes of clinical signs into a host of unimportant particularities, as if a correspondence existed between the symptoms of the visible alteration—the tissue reaction and the outward appreciable change in function (the *extrinsic* work)—and the alteration in the *intrinsic*, invisible work of the tissue, the amount of force produced, and the amount of energy expended,—in other words, the *output*.

When the new methods of diagnosis, auscultation and percussion, yielded such remarkable additions to our knowledge of the general symptoms by disclosing the local conditions, it was believed that prognosis and treatment might be directly deduced from the physical changes. The utter failure to recognize the important fact that a physical explanation of the phenomena implies a thorough knowledge of dynamic and biologic processes, the relation between *visible* work or reaction, and *invisible* work, resulting in the production of energy, naturally led to the erroneous belief that the changes in volume, consistency, and power of motion of the tissues and organs appreciable by physical means, the condition, in short, which we term tissue disturbance, functional anomaly, or *symptom*, was identical with the intrinsic processes of energy production. But a simple consideration of the actual facts teaches that the visible work can only correspond to an equal change in the equilibrium when the power of reacting, the original irritability, the relation between nervous discharge and work production, remain the same. To attempt to judge of the amount of energy used up and available in the organism by the degree or kind of reaction present would be about as erroneous as to attempt to deduce from the loudness and nature of a report the strength of the discharge or the nature and size of the substratum which produces the sound-waves or even the number of possible detonations.

After a period of unbridled enthusiasm the observations and critical analyses of impartial investigators, as was to be expected, gradually reduced our methods of diagnosis to their true value and showed the difficulty of arriving at a correct judgment in regard to changes in function by a mere consideration of certain acoustic phenomena. The conviction was gradually reached that none of the methods afforded pathognomonic signs, that is to say, such as permitted a simple and direct conclusion, and that the sign in itself is of secondary importance. It is the combination of all the signs, and the synthetic and analytic ability on the part of the observer, that are required to produce the true clinical picture, just as the perfect understanding of a thing or process depends on the number of essential qualities known and the power of uniting them into a harmonious whole.

In spite of all this, in spite of the periodic oscillations from the extreme of optimism to the extreme of pessimism in regard to the latest method, which to the student of medicine is merely the milestone of a new epoch, new methods are constantly being promulgated and greeted with the same enthusiasm. The search for the pathognomonic symptom has never been abandoned, and we still hope to solve the riddle of energy production and conversion and of the primary nervous discharges in the old way and to deduce the changes in the intrinsic work from the results of extrinsic work, the visible reaction, although in the various forms of reaction only a very small part of the coarsest changes become manifest. In a word, the visible work, measured by the energy required to perform it, is no more than the final account of the *expenditure* in *vital force*, the individual items of which are just as much hidden as are the amount of the reserve fund and the individual items of the *income of vital* energy (by waves of light, heat, sound, and other waves that reach the organs of sense).

Again, it was thought that the veil was about to be lifted when bacteriology promised to solve the mystery of the origin of functional alterations, in other words, to isolate the long-looked-for etiologic unity, the primary cause or irritant. The conviction that it was possible to find a definite cause for disease promised to satisfy all the demands of the physician, and thus the demonstration of certain forms of micro-organisms appeared to solve in the simplest way the problems of diagnosis, prognosis, and treatment.

This hope, however, was again nipped in the bud by impartial clinical observation, and it now appears that the pathognomonic symptom existed only in the imagination of those who cannot and will not comprehend that the alterations in the internal work (the production of the forces which accomplish the cohesion of the organism) are only imperfectly manifested in the sum of individual reactions which we call disease or morbid process. To determine by the kind and amount of this extrinsic work—the visible symptoms—the amount of energy stored up which makes possible the conversion of the vital forces, and hence the maintenance of the machine, is no more possible than it is to determine the specific heat or capacity for heat of a body by the amount of heat radiating from it. Unless the specific heat is known, neither the amount of heat present nor the amount of work that has been or can be performed can be ascertained. Unless we know the *disposition,*—that is, the excitability of the organism,—we cannot estimate the significance of a single output or symptom.

Since, then, the visible reaction is the result of invisible causes, and the invisible ability to develop energy, or, to use medical terms, since the symptom merely represents the product of irritant and disposition, the solution of this equation with two unknown quantities must necessarily yield an endless variety of answers. The same irritant with a different disposition will produce as many different forms of reaction as the same disposition with different irritants.

Since, therefore, what we recognize as reaction requires only a part of the energy present, and merely represents that part of the process which can be appreciated by physical means,—just as it is impossible to deduce from the amount of radiating heat the degree and kind of the process which produces the heat, or to deduce from the amount of work performed the conversion and ultimate capacity for work of the processes which produce the energy of motion,—the result of a single method of investigation, the form of reaction, or the individual symptoms can be utilized as a basis for judging an individual case only when the amount of local and general disposition or the intensity of the irritant is known.

The same form and an equal number of micro-organisms must produce different symptoms and different clinical pictures, according to the disposition, just as different forms and numbers of micro-organisms may produce the same phenomena if there is a corresponding variation in the power of reaction. During the era of injection with tuberculin a good opportunity was afforded for studying the differences in reaction produced by differences in disposition; the dose of the irritant (the tuberculin) could be regulated at will, and the extent of the reaction, and especially the febrile reaction, could be carefully determined.

Hence it is not the finding of certain species of micro-organisms, but the extent of the irritability by the foreign bodies and their products,—in a word, the disposition,—that is the essential point both in making the diagnosis and in laying down the treatment; but the disposition can only be judged by clinical means. The demonstration of micro-organisms is not without a certain value. Thus, for instance, the presence of tubercle bacilli in the sputum proves that there is a certain loss of resistance in the pulmonary tissue indicating the probability of further tissue changes and furnishing evidence that the soil is also favorable for the growth of pus-producing micro-organisms.

But can the different species of micro-organisms be utilized as a basis for classification without at least the same limitations that are necessary for other irritants? A system reared on such a basis is at best an artificial one. A natural system can be built up only on the basis of functional diagnosis and by taking due account of the various amounts and forms of energy that enter into the consideration. But, hampered as we are by unnatural classifications and the lack of proper methods for measuring the amount of essential work performed, we are still far removed from such a method of studying disease. We content ourselves with regarding the forms of disease as permanent phenomena, or conditions to be permanently fixed by a more or less descriptive appellation; as if a satisfying knowledge of the process could be arrived at by such a method of labeling, whereas, as a matter of fact, we are simply confining the countless variety of processes constantly going on in the body within the narrow bounds of the systematic text-book.

To develop these points of view as fully as they deserve at this beginning of a new epoch in medicine would carry me beyond the limits of the present article. I wish merely to point out how difficult it is to judge of the process in the healthy and in the diseased organism if we regard health and disease merely as special manifestations of energy in the organism and not as the object of scholastic definitions, which, starting from a non-essential standpoint, are necessarily vague, and have already lost their value, since they do not reproduce the vital processes, but are mere abstract ideas intended to satisfy the need of the human mind for generalization.

Medicine will always be the art of deducing correct conclusions in regard to the energy-balance of the body from a number of facts collected from different quarters and different points of view. The experience of centuries cannot be discarded, and we constantly return to the ideas which have stood the test of time and have shown themselves to be at least approximately true.

Among the most trustworthy results of experience are the importance of disposition and the fallacy of pathognomonic symptoms.

Any one who attempts to judge a complex of processes which we call disease by a single symptom or the result of a single method is as much in error as he who would attribute a certain degree of temperature of a body to the combustion of a certain quantity of coal, whereas both the forms of heat-production and the amount of radiating heat are subject to wide variations.

It is to be hoped that the time is not far off when it will again be recognized that those who regard only the local condition or a pathognomonic symptom are as much in error as those who take account only of the general morbid condition, and accord to a single (general) symptom—such as fever, for instance—an absolute value as a criterion in judging of the prognosis and planning the treatment.

The most vigorous patient may be destroyed or at least greatly injured by a purulent or a putrid exudate, if local diagnosis by means of exploratory puncture or operation were to be delayed because the general symptoms did not appear to indicate the necessity of speedy intervention, because the general condition was tolerably good, and the temperature approximately normal.

Just as little can the true state of the patient be determined by a mere investigation of the local condition, for even a small exudate may be extremely dangerous, and a large one relatively harmless. Many a purulent or putrid exudate in the hands of an experienced physician will end in recovery sooner than a serous exudate which has laid the foundation for a chronic process in the lungs.

The highest medical art demands that the general condition be not forgotten in the search for local changes, and, again, that the proper relations between the recognizable local conditions and the general reaction be determined by what is known in regard to disposition. A temperature of 40° C. (104° F.) in a child is often less significant than a slight chill in an adult, and it is well known that a clouded brain in children often means no more than a profound sleep in an adult.

It is the part of true medical insight to regard the symptoms not as the true nature of the disease, but merely as an imperfect picture and index of the alterations in the intrinsic work, and to determine the extent of disposition by carefully observing the effect of various irritants; for a knowledge of the disposition is absolutely necessary to determine whether the reaction is sthenic or asthenic.

II. HISTORICAL.

Inflammation of both layers of the pleura, also called **peripneumonia,** was not always recognized as an independent disease by classical medical writers of the eighteenth century, notwithstanding the fact that in the writings of Hippocrates it had already been accorded an independent position, at least so far as the suppurative form is concerned. Sydenham,[1] * for instance, regarded pleuritis and pneumonia as expressions of a blood disease becoming localized either in the lung or in the pleura. Boerhave appears to have been the first to attempt an accurate distinction between pleuritis and the diseases of the pulmonary tissue.

Although numerous observations on the treatment, course, and termination of purulent pleurisy are found in the writings of the older authors; although Baglivi[1] described a form of latent pleurisy, and Auenbrugger, the talented founder of percussion, had contributed several important diagnostic signs based on percussion, it may nevertheless be said that the history of **pleurisy** as a new and independent nosologic species dates from the time of Laennec's[1] investigations. This great clinician has left us the frame and chief lines of one of the clearest pictures that have ever been given, and, notwithstanding a number of more or less essential additions, it maintains its position to the present day.

The great progress in pathologic anatomy favored the belief that the visible alterations in the tissue observed at the postmortem table represented the cause and true nature of the disease, and led to the hope that a further perfection of the so-called physical methods of examination, based on pathologic anatomy, would reveal the actual character of the different alterations in the tissue. As a natural result of these efforts and of the great variety of morbid symptoms observed, new divisions of the morbid process, based on signs which were frequently unessential, were constantly made without due regard to the alterations in the function and other important physiologic processes.

The earliest advances are marked by the work of Wintrich,[1] which still remains a mine of experimental and clinical observation and a pattern of the most scientific method of clinical investigation, inasmuch as it recognizes all the various methods as equally legitimate, but not necessarily equally valuable. Later Skoda[1] and Traube,[6] the value of whose contributions to the science of physical diagnosis is indisputable, added their quota to the symptomatology of pleurisy.

In some of the more recent experimental and clinical studies the attempt has been made to investigate the effects of *pressure, circulation,* and *absorption* in the pleural cavity and in the lung, as modified by the presence of exudates, and to determine the chemical composition of the exudates themselves so as to obtain new data for the proper interpretation of individual conditions and the appropriate medicinal treatment.

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* The small figures throughout text refer to the various subdivisions of the Bibliography, beginning on page 962.

Among these are to be mentioned the work of Ferber,[6] who has given a minute description of the *physical signs;* that of Garland,[5] which refers particularly to the *mechanical problems* involved; and that of Lichtheim,[5] who studied the *changes in blood pressure* produced by occlusion of the pulmonary blood-vessels. Leyden,[5] Schreiber,[5] and others made careful manometric investigations on their patients. Rosenbach[5] has contributed a critical discussion of the experimental work on changes of pressure in the pleural cavity, in which he analyzes the results of a long series of experiments and clinical observations.

During the past decade the *bacteriologic* aspect of the question has been carefully worked up, especially in the writings of A. Fraenkel[5] and Netter.[5]

The tremendous advance in surgical methods during the past thirty years led to the publication of an extraordinarily great number of articles devoted to the *operative treatment of pleurisy.* The greatest advances in this field are coupled with the names of Trousseau,[1] Traube,[6] Kussmaul,[12] Dieulafoy,[12] Bowditch,[12] and Bülau.[12] The method of operating by means of simple thoracotomy, reserving resection of the ribs for rare cases, found its principal advocate in Rosenbach,[13] who was also the first to advise against the use of irrigation in the after-treatment and to recommend the use of iodoform.

Two monographs—that of Fräntzel,[1] published twenty years ago, and Gerhardt's[1] paper, which appeared recently—are remarkable for their wealth of clinical experience and contain a most instructive survey of the state of our knowledge and the fluctuation of opinions on the most important questions.

In concluding this short historical review, we may mention as the most important advance in diagnosis during recent times the introduction of *exploratory puncture.*

The introduction of this method, which we owe to C. A. Ewald more than to any one else, marks a new epoch in the diagnosis, prognosis, and treatment of diseases of the pleura, and although it must be emphasized that exploratory puncture, like all other methods, is not always absolutely conclusive, it is, nevertheless, in the hands of a skilful and experienced physician, the only method of procuring the material necessary for arriving at a definite conclusion with rapidity, and at least comparative certainty, in cases where the whole array of ambiguous symptoms obtained by the older methods would not suffice to guard against error.

III. PATHOGENESIS AND GENERAL ETIOLOGY.

Inflammation of the pleura, pleurisy, or **pleuritis** (in French, *pleurésie*), is, in its milder forms, one of the commonest of all diseases. Changes in the pleura belong to the commonest alterations found at autopsies. Any variety or grade of inflammatory product, from a delicate "breath-like" opacity and slight, barely perceptible deposit, to solid plates an inch in thickness and covering the entire pleura,—from flocculent, irregular adhesions that are readily torn to a dense formation of connective tissue completely gluing together the entire pleural sac,—from a few drops of limpid fluid to many liters of purulent or putrid contents,—from the faintest, barely perceptible ecchymosis to an extensive hemorrhagic exudate have been observed.

Interesting as the wide variations in the tissue changes may be to the histologist, their interest to the clinician and practising physician must necessarily be restricted, for the functional disturbances depend on a wholly different array of factors, and the same histologic changes do not always possess the same clinical significance, or at least are only found associated with the same symptoms. For this reason I shall not attempt a special description of the morbid changes according to the classification commonly found in text-books on pathology. The histologic changes will be described only when they afford important data for diagnosis and treatment.

Age and Sex.—Changes in the pleura occur during embryonal life, and are quite frequent during the first years after birth. Sex does not appear to exert any influence; in extreme old age a terminal pleurisy, barely distinguishable from hydrothorax, is almost as common an occurrence as hypostatic pneumonia.

The contention of a few authors that pleurisy affects chiefly the **left side** is not confirmed by my observations. It may be the case in certain forms of tuberculosis, but the difficulties of obtaining reliable statistics on the question are obvious, as it is impossible to determine at the autopsy whether, in an apparently normal pleura, a mild or even a severe attack of pleurisy may not have been present without leaving a trace, and, on the other hand, investigations pursued on the living subject may fail to reveal a number of morbid conditions on account of the mild nature of the symptoms.

In organic hemiplegia, after cerebral disease, O. Rosenbach has shown that the paralyzed half of the body is almost exclusively the seat of pleural or pulmonary disease. He attributes this either to a diminution in reflex activity of the paralyzed side, or to trophic influences associated with the decay of cerebral innervation. It is a well-known fact that both the reflex and the voluntary portions of respiratory activity in such cases are much weaker in the paralyzed than in the sound half of the body, both immediately after the insult and for some time afterward.

Localization.—Owing to our deeply rooted craving for a localizing diagnosis, we speak of double, pericardial, diaphragmatic, mediastinal, apical, and intralobar pleurisy (see page 894).

So-called "occult pleurisy" formerly enjoyed considerable reputation. It cannot, of course, be regarded as a special variety, and merely indicates the incompleteness of our methods of diagnosis, a certain superficiality on the part of the examiner, or a diminished reaction in the patient.

Double pleurisy is frequently observed in experiments on animals, but comparatively rarely in living subjects, probably because the healthy pleura of an animal, after forcible introduction of relatively large masses of material calculated to produce inflammation or suppuration, offers conditions of absorption and irritation very different from those of a pleura that has become diseased in the ordinary way. It may be assumed in general that in this case the irritation is so slight in proportion to the amount of actual resistance that every means of protection is called into play, so that the irritative material is prevented from spreading beyond the point originally attacked.

Double pleurisy is relatively frequent in systemic and in infectious diseases, in tuberculosis, carcinoma, sepsis, pulmonary infarcts, and after double pneumonia.

Pericardial pleurisy, which obviously may have its seat in any portion of the outer surface of the pericardium bounded by pleural tissue, assumes a serious aspect on account of the—very much exaggerated—danger that the process may suddenly extend to the inner surface of the pericardium and to the endocardium. Correct early diagnosis in such cases is important from a prognostic point of view, as internal pericarditis, which has a number of symptoms in common with pericardial pleurisy, is unquestionably a much more serious disease.

In the same way *diaphragmatic pleurisy* possesses special clinical importance because even when the inflammatory process is relatively limited it produces apparently dangerous symptoms, such as cyanosis, air hunger, and a certain condition of stenocardia, partly because of the severity of the pain and partly because of the mechanical hindrance to diaphragmatic breathing which is always present, being due to the edema and acute infiltration of the muscle.

This affection, as the subsequent course usually shows, is far less dangerous than it first appears to the observer and to the patient himself. It presents much the same appearances as those observed in persons suffering from simple painful rheumatism of the thoracic muscles, in whom the interference with respiration may, until the pain has been relieved by injections of morphin, bring about a very alarming dyspnea which may easily be confounded with cardiac weakness or congestive pulmonary edema, although the pulmonary tissue itself retains its integrity.

The distinction between **primary** and **secondary pleurisy** is justifiable on theoretic grounds. In practice, however, the distinction is exceedingly difficult and often impossible, although it cannot be denied that a positive distinction between the two forms may be of the greatest importance both for the treatment and for the prognosis. At the very outset it is important to recognize that the disease is primary—in other words, localized in one spot which has been attacked by the sum of all the insulting factors, or has borne the brunt of the attack single-handed. We may, of course, disregard cases in which we have to deal with the local effect of a trauma or with a single injury to the organism which is not of a nature to become more extensive. If the primary nature of the disease can be established, the danger of its becoming general or resulting in a multiple localization may in most cases be excluded; the pleurisy will in all probability remain a local disease. At the cost of the internal activity of a single organ, which we term inflammation, the attack is diverted from all the other organs. Even micro-organisms may under such conditions—that is to say, when, during their passage through the tissue-juices, they become arrested in a single organ as in a sieve—give rise only to an intense *local* disturbance, whereas, if they were to settle in various localities, they would produce a multiplicity of disease foci the consequences of which could not be foreseen; as, for instance, when a deleterious substance penetrates to the interior of an important organ instead of becoming permanently arrested in the investing serous membrane.

The view here presented may seem strange to many who, on experimental grounds, believe in a uniform distribution of all foreign bodies entering the circulation. It must not be forgotten, however, that the conditions in disease are somewhat different, since the morbific agent reaches the general circulation only through the venous and lymphatic channels, and usually very gradually, after certain changes have taken place; whereas, in experimental work, enormous masses enter the blood-stream at once. Now, the serous membranes appear to possess the special faculty of arresting abnormal substances and micro-organisms, and restraining them from entering the essential tissue of the organ. Even in the case of poisons which may produce a general disease—as, for instance, the poison of acute articular rheumatism—the intensity of the local disease is, as a rule, inversely proportional to the number of disease foci. When one joint alone is attacked, suppuration may supervene; but when several are attacked at once, they each present only the signs of serous inflammation.

Now, as the various foci do not, as a rule, develop at the same rate—since the difference between the power of individual tissues to react and the intensity of the irritative agent being so great that a poison which reaches different points of the body at the same time calls forth a noticeable reaction only after variable periods of time—it is beyond our power to decide at once in a disease which appears to be primary, that is, a disease in which by our methods of investigation there appears to be but one localization, whether other manifestations may not appear at different places within the next few days or weeks, the germ having been planted at the same time but developed relatively more slowly.

If, for instance, in rheumatic polyarthritis, one joint after another or the serous surface of the pleura and of the heart are successively attacked, it is not necessary to assume that the poison has been carried from the joint which was originally involved, and that the other localizations are therefore secondary. On the contrary, we may, and in many cases must, consider all the manifestations as of equal significance, and regard even the pericarditis and pleurisy which follow in the wake of articular affections as manifestations of the general disease.

All the disease foci are accordingly regarded as localizations, as the expression of the rheumatic infection or poison, and therefore represent an identical primary affection. It is true that if we conceive the entrance of the poison into the blood or the so-called systemic disease, the localizations of which are usually not demonstrable because so slight and so widely diffused, to be the primary affection, we must restrict the term primary localization to cases where no general disease can be demonstrated and the infection results in an immediate localization in one organ.

Since, however, the morbific agent of polyarthritis is not the disease itself, and articular rheumatism is not an entity, but simply a term chosen on account of the principal localization and applied to certain visible alterations in the function of the articular serous membrane directly referable to a definite irritation, so the reaction of the pleura or of the pericardium is simply an increase in its function due to the activity of the irritative agent, or to its special localization, or to predisposition.

It follows that the pleurisy of rheumatic polyarthritis, which is not always identical with true rheumatic pleurisy, must be regarded as an inflammation equal in rank to the usual localizations of articular rheumatism in the joints, especially when it appears comparatively soon after the period of articular inflammation; whereas pleurisy occurring as a late sequel is due to some change in disposition, and should be regarded as a secondary disease.

Again, the form of pleurisy following in the wake of a distinct disease of the throat or other organic trouble may with every right be termed secondary. Even articular rheumatism following an unmistakable attack of inflammation of the throat is to be regarded as a secondary affection because the disease of the pharyngeal organs simply prepares the soil for the reception of a special poison distinctly different from that which produces the throat affection.

The more independent the primary throat disease in point of time, character, location, and extent, and the more the general condition of the patient is affected by it, the less are we justified in regarding other localizations as primary infections of equal rank; whereas, disturbances in the joints or other inflammations occurring simultaneously with the throat affection may with propriety be regarded as similar manifestations of a relatively benign or malignant general disease. The disease which, from its principal localization, is known as *acute articular rheumatism* we have every reason to regard as the mildest form of the group of tissue changes characterized by the development of *pyogenic micro-organisms*, the highest grades of which are represented by the pyemic affections (O. Rosenbach).

It would appear from these considerations that classifications of this sort can have no practical value, since the clinical picture, the order of succession, and the number and variety of localizations depend not on the nature of the poison, but on the co-operation of irritant and disposition or irritability, and the disposition itself may, under the influence of an irritant and under certain conditions, undergo various alterations.

Rather than attempt to draw the line on strictly genetic grounds, it will be more in accord with the needs of practice to regard as secondary pleurisy those forms in which the local changes in the tissue activity of the pleura which we call inflammation, and the general and local functional disturbances depending on such changes, are overshadowed by a basal disease which, even without implication of the pleura and without the occurrence of inflammatory or febrile reaction, would produce a marked disturbance in the body economy; and, secondly, those conditions in which permanent or temporary disturbances originating outside of the pleura create favorable conditions for the action of relatively slight—*i. e.*, secondary—poisons in the domain of the pleura.

In the former case the abnormal tissue process is at the same time the exciting agent, and the expression of an altered disposition; in the latter case two different tissue processes often exist side by side, one of which furnishes the irritant, while the other effects a modification of the irritability—in other words, a change in disposition.

To this group belong the pleurisies which depend on constitutional diseases and present themselves under the form of a so-called mixed infection or as a sequel to the general disease in previously weakened individuals. They include *terminal pleurisy* and certain forms of *subacute* and *acute pleurisy* without any tumors in the tissues of the pleura, occurring in tuberculous and carcinomatous individuals; and, finally, *post-pneumonic exudative pleurisy* developing a few days after the termination of pneumonia by crisis or lysis.

The distinction between **acute** and **chronic** forms ought to be discarded altogether, at least for purposes of diagnosis, as it frequently happens that cases with a stormy onset subsequently develop into extraordinarily chronic processes, and, on the other hand, effusions which develop quite slowly and imperceptibly may terminate quite favorably.

Since, as we have shown, the initial symptoms and subsequent course of the disease are regarded merely as different modes of reaction of the tissue, depending on the relation of the irritant to the disposition, and as they cannot, therefore, be proportional to one of these factors, which may vary during the time of observation, it follows that the strength of the irritant can rarely be deduced, with anything like certainty as regards diagnosis and prognosis, from the intensity and extent of the initial symptoms. As a rule, both the severity of the irritant and the extent of the disposition are equally unknown. Even when the disposition is known to the physician from former observation, it is often so modified by antecedent bodily and organic influences tending to weaken the constitution by overexertion, by repeated illnesses, or even by the treatment adopted in the beginning of the attack, that the vigorous reaction of the initial stage is followed by an asthenic course, while, on the other hand, an asthenic beginning may, by appropriate treatment and the effect of adaptation, eventually lead to sthenic reaction or even to a crisis.

In any case even a skilled physician will require a certain length of time to determine a patient's disposition or susceptibility if he relies on physical examination and functional tests; and this knowledge is absolutely necessary to enable him to determine what even then is quite difficult: whether the process is to be acute or chronic. It must not be forgotten that the susceptibility of individual tissues may vary greatly both for the same and for different irritants.

The distinction between **diffuse** and **circumscribed** pleurisy is of no practical value, since the appearance of many small foci, separated from one another by considerable areas of sound tissue, in nowise differs from a diffuse process even when the extent of the individual foci is relatively small. Nor must it be forgotten that the physical signs are often out of all proportion to the true extent of the inflammation. Auscultation and percussion often yield positive results only in circumscribed areas, although the inflammatory process may extend far beyond such regions, and, on the other hand, it may also happen that the results obtained by one or all of the methods of physical examination are especially marked in regions where the pathologic changes are faintest.

The distinction between **dry pleurisy** (*sicca fibrinosa*) and **pleurisy with effusion** (*serosa* and *serofibrinosa*), which at first sight appears such a practical one, cannot be carried out at the bedside in the strict sense of the definition. In almost any form of pleurisy a few drops of serum can in the beginning of the attack be obtained by exploratory puncture. Even when pleural friction is obtained over more or less extensive areas there may be a considerable quantity of serum between the two layers of the pleura. Its presence does not always prevent the friction from being heard, as, depending on the thickness of the exudate and the position of the fluid, a number of points may remain where the two surfaces of the pleura can come in contact. Again, a friction sound must necessarily be produced when a solid exudate projects above the surrounding fluid like an island in the sea; or, in other words, when the fibrinous layer is thicker than the layer of fluid.

Of all the classifications, there is only one which in our estimation is of any value for clinical purposes. It is the classification according to the nature of the pleural contents as determined by exploratory puncture. Even this proposition, however, is contingent upon the proviso that exploratory puncture be used simply as a method, the application of which requires careful study and from which the observer does not necessarily expect specific or pathognomonic results.

For the adherents of the doctrine of bacteriologic diagnosis to the exclusion of all other means, a group to which the author does not belong, exploratory puncture carried out with the customary precautions affords the only sure basis of determining the variety of microbes present in the pleural contents. It would certainly be the only certain method of determining the etiology if the determination of one or more forms of microbes were synonymous with the determination of the causes or producers of the disease.

IV. SPECIAL ETIOLOGY.

Since, as has been mentioned, we take the stand that the functional and structural (visible and invisible) reaction called disease is a product of irritant and irritability (disposition of the tissue), two explanations present themselves for the origin of the inflammation. The pleurisy may develop because of the action of a maximal irritant on the pleura causing a maximum of internal tissue activity at the expense of the amount demanded by the needs of the organism, or because of some alteration in the disposition or irritability of the tissues producing an abnormal functional activity (inflammation) in response to an irritant which, under normal conditions, would not be sufficient for its production.

If there is any alteration in the internal (invisible) activity or energy necessary to be developed for the preservation of the organism, on which the degree of irritability—the power to perform visible work, in a word, the working capacity of the machine—depends, even a normal irritant acting on the diseased area will produce a variation in the amount of visible work performed. This variation we habitually term abnormal or morbid, and, if it is combined with permanent structural alterations of the tissue, it constitutes a tissue disturbance susceptible of anatomic demonstration.

Exposure to **cold, traumatism,** and **local infection** may be considered either as irritants (inciting processes) producing an alteration in the internal work and development of energy, the ultimate expression of which is inflammation, or as causes of an alteration in susceptibility sufficient to produce the local disposition necessary for an otherwise ineffective irritant to exert the special influence just described. They may accordingly, if of sufficient intensity, be regarded as the cause, or better perhaps as the inciting process, of the primary pleurisy; or they may be concerned in the production of a local (secondary) pleurisy only in so far as they modify the irritability and furnish a disposition (possibility) to an abnormal reaction. The reaction, however, does not necessarily occur if the necessary irritant or inciting process for the reaction, which under special circumstances is markedly modified, is absent.

Powder and dynamite have a different irritability, but the magnitude and variety of inciting processes which cause the visible reaction —the explosion—are quite as different as the internal processes on which this reaction depends.

In other words: if in two individuals exposure to cold, traumatism, infection with some noxious agent, or any other influence produces an alteration in the irritability of the pleural tissue, one of these individuals may, under the influence of some additional agent,—such, for instance, as a fresh exposure to cold which under other circumstances would not be sufficient to produce any alteration in the internal work,—at once manifest the symptoms of an acute or subacute inflammation of the pleura; while the other, after suffering a few days of impaired power of resistance (power of reaction—*relative good health*), may gradually, either spontaneously or under the influence of appropriate measures, such as rest in bed, etc., regain his former power of adapting himself to ordinary irritants, *i. e.*, *absolute health.*

Accordingly, the **causes of pleurisy**—in other words, the inciting processes and the influences which determine the disposition necessary for such processes to become effective—are very numerous.

Pleurisy, in which the last irritant is regarded as the sole cause, though it is often only the accidental inciting process, occurs:

1. As a frequent sequel and concomitant of almost all constitutional diseases and profound disturbances of nutrition. In these cases it follows from what we have just said that it is, as a rule, impossible to demonstrate the true insult and the direct cause.

2. During the period of convalescence from any protracted disease or after loss of blood. This form is usually associated with thrombosis in the veins of the lower extremities.

3. In all acute and infectious diseases, especially in all forms of endocarditis and pyemia (puerperal fever), and in acute articular rheumatism which probably represents the mildest member of the group of general diseases characterized by the presence of pyogenic organisms.

4. In ulcerations of adjoining regions, the ribs, the esophagus, the stomach, the mediastinum, the vertebræ; especially in inflammation of adjacent serous membranes, the pericardium and peritoneum, in which case extension occurs by way of the larger lymph-channels or through the parenchyma.

5. In all forms of acute and chronic inflammation of the lungs; in purulent, putrid, or caseous degenerations, especially when the diseased focus is superficial. In diseases of the bronchial mucous membrane pleurisy can almost always be excluded.

6. In chronic nephritis, in degeneration of the heart-muscle, and cirrhosis of the liver, pleurisy with effusion resembling the product found in hydrothorax is very common (see page 885). When the two last forms are present, they are almost always on the right side, and constitute a very grave complication, presaging a fatal termination.

In ill-nourished and aged individuals pleurisy is very frequently the *terminal* disease. In all inflammations within the peritoneal cavity, especially in perityphlitis, the pleura and the subphrenic space must be subjected to a careful examination, as serous or purulent pleurisy is one of the most frequent *complications* and occasions many difficulties in diagnosis and prognosis. For, if a pleurisy of this kind escapes detection, as, for instance, when it barely extends beyond the surface of the diaphragm, a continuance or exacerbation of the fever is naturally attributed to an aggravation of the primary peritoneal affection unless the subsidence of the local symptoms is so clear as unmistakably to suggest the presence of a new disease. Similar difficulties in diagnosis may, of course, present themselves in primary pleurisy if it is complicated during the stage of regeneration by a hidden suppurative peritonitis.

It is rather remarkable that pleurisy is so seldom observed in typical cases of *influenza*, because bronchial and pulmonary disease is particularly common in this condition, especially on the left side.

Again, in *typhoid fever*, and particularly in *measles*, both of which are frequently accompanied by acute and chronic pulmonary disease of varying degrees of intensity, serous and purulent pleurisies are comparatively rarely met. Pleurisy is, however, an occasional sequel of typhoid fever in debilitated patients.

The relations between pleurisy and *tuberculosis* demand special mention in a section on etiology. There is no doubt whatever that so-called genuine pleurisy is very often an early sign of a tubercular predisposition, or even the expression of minute localizations of the disease in the lungs or bronchial glands. The relations between these two conditions have received especial attention in the most recent literature, and in a series of statistics von Barrs,[2] Coston,[2] Dubville,[2] and others have made a fresh attempt to prove that pleurisy is always a trustworthy symptom, or, more correctly, a sure forerunner of local or general tuberculosis. The domain of traumatic etiology has also been encroached upon by Netter,[4] who claims that in 68% of the cases attributed to traumatism tuberculosis must be regarded, if not as the cause, at least as the predisposing factor in the production of the disease.

My own experience leads me to regard these views as too radical, for although I must agree with Fiedler[2] in regarding many forms of pleurisy as mere localizations of a rheumatic or pyemic infection, and believe that a great variety of infectious diseases, from simple pharyngitis to the severest form of pyemia, may lead to localizing symptoms in the pleura in the entire absence of any tubercular etiology, it must nevertheless be admitted that most of the cases accepted in the statistics on the strength of postmortem findings are not absolutely conclusive, because of the difficulty or even impossibility of proving any connection between two events when a long interval of time has elapsed between them.

In constructing an etiologic chain of this kind one must bear in mind, *à priori*, the possibility that a pleurisy acquired as a result of articular rheumatism may, without any connection with the basal disease, later be followed by the development of a tuberculous affection, just as, conversely, a pleurisy the remains of which many years later fail to show any trace of tubercle bacilli or their products may have been tubercular in character from the outset, for we know from experience that the healing of tubercular lesions is particularly apt to be followed by marked alterations in the connective tissue which bear testimony to the former presence of extensive disease.

It is quite true, as Hanau[2] assumes, that in such cases calcification of the bronchial glands must be demonstrated before one is justified in interpreting the original disease as tuberculous. It must not be forgotten, however, that calcification may constitute the terminal stage of any form of marked and persistent swelling; in other words, of so-called chronic non-tubercular inflammation.

It is evident, therefore, that we cannot exclude a tuberculous etiology in cases of old pleuritic conditions when the pathognomonic signs of tuberculosis are absent, any more than we can say definitely that any pleuritic adhesion associated with marked pulmonary tuberculosis is actually of tubercular origin.

For this reason the statistics of Schlenker,[2] which in many respects deserve much credit, are not quite conclusive, as the argument of a tubercular etiology is based solely on the coexistence of pleuritic adhesions with the signs of gross tubercular disease of the lungs. For, although it is true that in 33 out of 57 cases of pleuritic adhesions the presence of tubercular disease of the lungs was demonstrated by the microscope, yet this proportion, remarkable as it is, fails to explain either the temporal or the causal relation between the two manifestations.

We are therefore forced to the conclusion that the coexistence of tissue changes in the pleura with tubercular localizations elsewhere does not suffice to prove either that the pleurisy was caused by tuberculosis or that a former pleuritic affection was of a tubercular nature. While, on the one hand, pleurisy may induce tuberculosis by the permanent functional disturbance which it produces in the lung, and, on the other hand, tuberculosis of the lungs may, by the associated irritation of the pleura, be the cause of pleurisy, yet the two processes may exist quite independently.

One thing only appears to be certain: The alterations in the respiratory mechanism and in the internal activity of the tissues produced by an imperfectly healed, non-tubercular pleurisy may bring about an alteration in the susceptibility of the tissue which becomes the true foundation for the development of tuberculosis in the pulmonary and pleural tissue, or, as the saying is, may furnish the soil for the growth and development of the tubercle bacillus. Notwithstanding the teaching of modern bacteriology, we cannot renounce the only possible standpoint for the physician, approved by the observation of centuries, that a certain disposition—or, in other words, a profound alteration of the invisible work of the tissues for the production of energy, known as *susceptibility* (the first foundation of visible activity or reaction)—furnishes the necessary conditions for the colonization and proliferation of micro-organisms which preferably either possess or assume the characters of the tubercle bacillus, but may also belong to one of the species of pyogenic micro-organisms (bronchopneumonia, mixed infection, etc.).

Nor can it be doubted that **tumors of neighboring organs or of the pleura** itself may act as the exciting agents of tissue activity and directly or indirectly produce a true pleurisy, that is to say, the form of alteration in the local tissue work of the pleura which we designate inflammation. For the nutritive disturbance caused by the tumor may, on the one hand, intensify the predisposition and enable an otherwise ineffective irritant to exert an abnormal influence n the domain of the pleura, just as, on the other hand, the growth of the tumor itself may act as an exaggerated irritant.

Tumors of the Pleura.—Carcinoma, sarcoma, enchondroma, endothelioma, and exceptionally even lipoma are observed in the pleura. The tumor is more apt to be metastatic than primary, being derived from neighboring organs, most frequently the mammary gland, the mediastinal glands, the liver, stomach, etc. Tumors within the pleural sac may also be due to the action of echinococci; these tumors have recently been made the subject of an exhaustive description by Maydl[3] with special reference to the treatment. I shall return to the discussion of this subject later on in connection with diseases of the lung tissue; the most important points in the diagnosis of this uncommon disease will be found on page 889.

Attention has recently been called by A. Fraenkel[3] to a special form of malignant disease of the pleura, so-called *endothelial cancer*, already described by E. Wagner[3] and others. The disease, which is regarded by various authors as the result of a proliferation of the endothelium of the blood-vessels, and in which the pleura occasionally presents the same appearance as a hypertrophied vesical mucous membrane, generally manifests itself under the form of a brawny pleurisy, the microscopic examination showing diffuse nodular and brawny adhesions between the two layers of the pleura frequently going on to a complete contraction of the tissue.

It has been shown by numerous microscopic examinations that the lymph-channels and the ground-substance of the pleura are equally concerned in the proliferative process. But there is still much difference of opinion in regard to the classification of the various forms of tumors. Neelsen[3] leans toward the theory of metastasis, and includes the processes among the infectious inflammations; Schottelius[3] regards the disease as a carcinomatous lymphangitis, Schweninger[3] as a proliferating lymphangitis.

According to A. Fraenkel, there is found in transverse sections of the costal pleura a uniform proliferation of the connective tissue with very few cells, within which the lymph-spaces appear greatly dilated without losing their linear cleft-like character, and more or less filled with polymorphous, cuboidal cellular elements, epithelial in character with vesicular nuclei. These cellular elements either occlude the lumen completely or invest the walls with one or more layers, leaving an open space of variable width. The phenomenon is most marked in the central portions of the pleura. In places there is found, instead of this accumulation of cells, a finely granular mass containing a few leucocytes. The connective tissue between the open spaces contains many spindle cells and numerous foci of round-celled infiltration. The endothelial covering of the pleura is for the most part lost, the denuded areas being covered with the already mentioned cuboidal, polymorphous elements. The lymph-clefts and the vessels of the diaphragmatic and mediastinal pleuræ are markedly dilated and filled with epithelioid cells, so that they closely resemble large cancer nests. Similar, though less pronounced, alterations are found in the lymph-glands.

I will venture to express the opinion that this form of tumor formation is not a primary process, but a secondary proliferation of endothelial and epithelial elements, and cannot therefore be regarded as a true malignant new-formation. This view is supported by the fact that there was no metastasis in the cases described by A. Fraenkel[3] and others,[3] the tissue alterations being confined to the pleura and external surface of the pericardium, whereas it is well known that malignant tumors (colloid cancer, carcinoma, and true endothelioma) are characterized by an extraordinary and rapid tendency to metastasis. I am therefore inclined to agree with Neelsen's[3] view that so-called proliferating lymphangitis does not represent the proliferation of a tumor so much as the ultimate consequences of an intense chronic inflammation.

An explanation of the peculiar tissue products may be facilitated by a more careful consideration of the processes at work in the development of **diffuse brawny** (connective-tissue) pleurisy.

The tissue alterations are produced (1) by a proliferation of the already existing connective tissue as well as of that which becomes organized from the inflammatory products; (2) by swelling and proliferation of the true endothelial elements of the pleura still remaining, the number of which, in advanced cases, may be exceedingly small on account of the rapidly progressing degeneration of the cells; (3) by extensive new-formation of vessels and proliferation of the epithelial elements in the vessel-walls both in the muscular and in the endothelial layers. This proliferation of endothelial and epithelial elements, especially the marked development of the characteristic polymorphous, cuboidal epithelioid cells, which I have observed particularly in cases of massive plastic pleurisy, appears to me to indicate a new-formation of blood-vessels and occlusion of the vessels going on hand in hand with the chronic inflammatory process, since the endothelium of the blood-vessels plays the chief part, just as it does in the formation of acute thrombi.

I have not been able to satisfy myself that the proliferation, or rather transformation, of epithelium into cuboidal cylindric cells is especially confined to the lymph-clefts. A study of a number of specimens of this condition has left me with the impression that we have to deal with an epithelial and connective-tissue new-formation very similar to that observed in hypertrophic cirrhosis of the liver and in the peculiar form of pneumonia following division of the vagus. In the former case the well-known accumulations of peculiar cuboidal cells, suggesting the appearance of gall-ducts, are found in great numbers. In vagus pneumonia we find within the thickened bands of connective tissue cross-sections of bronchi and blood-vessels more or less completely filled with the same cuboidal, polymorphous epithelial cells which unquestionably do not represent a primary tumor formation; they are rather the expression of a secondary proliferation of a lower order of tissue following degenerative atrophy of the true parenchyma.

The intensity of this hyperplasia of the blood-vessel endothelium must be the more pronounced in all cirrhotic processes,—that is, such as eventuate in connective-tissue proliferation with destruction of the essential tissue,—the more the blood-supply is increased by the morbidly exaggerated irritation during the true hypertrophic stage, and the more successful has been the attempt to compensate for the disturbance by the formation of new plexuses of blood-vessels. The degree of hypertrophy of the blood-vessel endothelium is, therefore, proportional to the original hypertrophy of the parenchyma which preceded the degenerative process; and the accumulation of epithelial cells is only to a very slight extent due to the proliferation of the nuclei or cells of the connective tissue or of the few remaining true parenchyma cells. The new-formation probably originates in every case not from the essential tissue but from the epithelial coverings of the more resistant endothelial and epithelial structures.

Accordingly, the proliferation of the bronchial epithelium and of the blood-vessels in the lungs stands out conspicuously among the thickened bands of connective tissue, the alveolar epithelium having been utterly destroyed. In the liver the degenerated liver-cells are replaced by the epithelial elements of the blood-vessels and gall-ducts. In the pleura, where there are no specific elements except the endothelium which rapidly disappears, nothing probably remains but an accumulation of endothelial cells from the blood-vessels, representing an abnormal new-formation of blood-vessels and giving rise to a variety of appearances indicating different stages of retrogressive metamorphosis.

V. BACTERIAL ETIOLOGY.

The interest of investigators during the past ten years has centered in the bacteriologic examination of pleural exudates. It is not too much to say, however, that this method of examination, in this as in other domains of medicine, has failed both in the attempt to give an indisputable explanation of the pathogenesis of pleural diseases in general and to furnish a means of determining the etiology with certainty in any individual case. Nor have the latest methods, so assiduously cultivated and greatly lauded for their practical value, succeeded in laying a foundation for any notable progress either in diagnosis or in treatment.

The fact, admitted by all authors, that fission fungi are absent in the great majority of serofibrinous exudates furnishes one of the best proofs that *the presence of microbes is not identical with the demonstration of the general or special causes of inflammations of the pleura.*

The assumption that the causal micro-organism of the inflammation cannot exist in the serous fluid does not afford a satisfying explanation for the negative bacteriologic result, if we consider that, in the first place, coagulated serum is a good medium for microbes in general, and, in the second place, bacteria are found in many cases even in the fluid serum.

In non-tuberculous purulent exudates various kinds of micro-organisms are almost constantly present, and the fact, already mentioned, that they are often found in serous exudates which do not contain cells conclusively proves that they do not owe their presence to the carrying power of round cells (phagocytes).

To reconcile these contradictions one is driven to the additional hypothesis that the micro-organisms make their way into the exudate only when they are not arrested in the interior of the tissue. But since the intercourse between the pleura and the contents of the pleural sac is exceedingly active, as shown by the absorption of all kinds of substances injected into the pleura, it does not seem probable that they should be arrested within the tissue, especially as the superficial elements are constantly being thrown off in any intense inflammatory process.

These considerations lead us to believe that a number of other irritants besides the micro-organisms are at work in the production of serous and fibrinous exudates, such as chemical bodies and physical irritants, the nature of which may or may not be known (heat, cold, mixture of the blood, etc.), and these irritants are not necessarily insignificant as compared with pus-producing micro-organisms, nor do they differ from them in quality.

In suppuration micro-organisms undoubtedly play an important, if not an exclusive and permanent, part; but it is equally certain that when the pleura has become predisposed in some definite way, the suppurative process may be kept up just as well by chemical substances the products of micro-organisms, as by the micro-organisms themselves. The fact that a sterile empyema is cured by merely providing a free outlet for the pus at least suggests the view that even sterile pus may contain substances which keep up suppuration.

The study of the micro-organisms found in the contents of the pleural sac—we advisedly do not say the etiology of pleurisy—has been much enriched by the works of A. Fraenkel,[4] Weichselbaum,[4] Netter,[4] Prince Ludwig Ferdinand of Bavaria,[4] Levy,[4] Jakowski,[4] and others.

In general, the results of bacteriologic investigations, on the authority of a work which Jakowski based on 52 of his own and 250 other cases, may be formulated as follows:

1. It is not always possible to demonstrate bacteria either in serous, or in purulent and putrid exudates; but before pronouncing the result negative, several attempts to cultivate the bacteria, as well as microscopic examinations and inoculation experiments, must be made.

2. If the bacteriologic examination is negative, serous and purulent exudates are to be regarded as *tuberculous*.

3. The majority of idiopathic, non-tubercular forms of pleurisy are caused by *Fraenkel's pneumococcus*.

4. In pleurisies following or accompanying pneumonia Fraenkel's pneumococcus is found more constantly than any other micro-organism; it is not rarely associated with streptococci and pyogenic cocci of various forms, even in cases that do not end in suppuration.

It appears, however, that exudates containing more than one species of bacteria, which are regarded as the products of a mixed infection, show a greater tendency to suppuration than the first-mentioned forms.

In purulent exudates the specific micro-organism of the basal disease, tubercle bacillus, typhoid bacillus, bacterium coli, etc., are frequently found associated with the so-called pyogenic micro-organisms. This form of inflammation must, of course, also be regarded as a mixed infection in which the true cause of suppuration remains in doubt. It is to be noted, however, that no incontrovertible proof has as yet been brought forward to show that the tubercle and so-called typhoid bacilli are pyogenic. [This statement should be modified. In 1887, A. Fraenkel * discovered the pyogenic properties of Bacillus typhosus, obtaining it in pure culture from an encapsulated peritoneal abscess. Experimentally this pyogenic action was first demonstrated by Roux,† and since by many other investigators. Cobzè,‡ by injecting the typhoid bacillus into the circulation of rabbits, and a few days later fracturing some of the bones, found that abscesses, from which the bacilli were recovered, occurred at the point of injury. While the suppurative conditions occurring in the course of typhoid fever are usually due to mixed infections, not less than twenty instances are on record where Bacillus typhosus was obtained in pure culture. In regard to the pyogenic action of the tubercle bacillus, it must be remembered that in the lung some lesions due to the tubercle bacillus may be distinctly inflammatory, and that a tuberculous meningitis may be largely purulent. Positive evidence of suppuration is furnished by Councilman,§ who described a purulent inflammation of the lung, and purulent infiltration of the intestine with abscess of the mesenteric lymph-nodes, due to the tubercle bacillus.—Ed.] *A priori* it may be assumed that these bacilli, like their products, may, in the presence of the necessary predisposition, be able to produce the most intense degrees of inflammation.

A few authors (Loniga,[4] Pensuti,[4] Fernet [4]) have attempted to show that even a serous pleurisy may be produced by the typhoid bacillus alone, or, to speak more correctly, that in the exudates formed in the course of typhoid fever, only those bacilli are found which are now designated typhoid bacilli, although they ought rather to be regarded as a subvariety of Bacterium coli commune developing under certain conditions. As in the majority of cases no bacteria of any kind are found in the serous exudates accompanying typhoid fever, there is not much ground for the assumption of the above-named investigators.

The large isolated cocci arranged in chains which Ehrlich [4] found in the malignant forms of puerperal fever, where he considers them a very unfavorable sign, probably do not possess the pathognostic significance with which they have been credited.

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* *Verh. d. VI. Cong. für innere Medicin*, 1887, 179.

† *Lyon Méd.*, 1888, No. 26, 1880. ‡ *Lo Sperimentale*, 1890, LXV, 651.

§ *Med. and Surg. Reports of the Boston City Hospital*, 1896.

It follows, therefore, that serous exudates do not, as has been stated, differ in any essential characteristics from other forms of exudates, for they not rarely, even in the entire absence of any tendency to suppuration, contain the forms of micro-organisms which are regarded as the specific pus-producers, while, on the other hand, empyema is often free from micro-organisms, and Fraenkel's pneumococcus is found both in serous and in purulent exudates.

In the empyema of tuberculous subjects there is often an entire absence of micro-organisms, and the presence of tubercle bacilli is so rare as to suggest an error of observation, or an accidental admixture, or the presence of a communication between a tuberculous focus and the pleura.

In examining sputum and pleural effusions containing tubercle bacilli in small numbers the bacteriologic examination, on which, according to the prevalent opinion at the present day, the prognosis is chiefly based, should be performed with unusual care, lest the presence of a few tubercle bacilli, which may have been deposited on the slide or in the staining solutions during former examinations, lead to serious mistakes in diagnosis. (See page 904.) [Inoculation tests should also be made.—Ed.]

It is difficult to explain the absence of tubercle bacilli in tuberculous exudates. They may have been completely destroyed by the overpowering mass of pyogenic micro-organisms or continue to exist in the form of minute spores that cannot be demonstrated, as in the caseous material found in tuberculous glands or joints.

There is not much in favor of the first view, since in a great number of cases of tuberculous empyema no pyogenic micro-organisms are found, which seems to show that the tuberculous exudate is not a specially good culture-medium for ordinary micro-organisms. Indeed, there is good ground for supposing that suppuration in these cases is produced and kept up not by micro-organisms, but solely by chemical products which the diseased tissue is unable completely to remove or expel, and which therefore keep up the irritation until the normal irritability is restored by the evacuation of the entire fluid containing the supporting substance of the irritant agent.

It is well known that the pleural effusion taken from tuberculous subjects and injected into the peritoneal cavity of guinea-pigs does not always produce tuberculosis, and attempts at cultivation and inoculation with portions of such exudates, even when the most appropriate media for the development of tubercle bacilli are used, are not always successful. In order to become a good nutritive medium for tubercle bacilli a sterile, coagulated pleural effusion must be treated before coagulation with about half the amount of veal broth and glycerin. At all events the bacteriologic behavior of exudates and empyema fluid taken from tuberculous patients goes to show that the numerous investigations in regard to the growth of micro-organisms have as yet failed to furnish a satisfactory answer to the important question of the relationship existing between tuberculosis and pleuritic alterations in which the physician is so vitally interested.

As a consequence of the exaggerated importance attached to infection as an etiologic factor, it has been erroneously supposed that *exploratory puncture* was the chief factor in the introduction into the pleura of carriers of infection. I cannot bring myself to accede to this view, since the accident cannot always be avoided, even when the most careful antiseptic precautions are taken, and clinical observation has long ago taught us that serous exudates, or, more correctly, serous exudates which already contain numerous pus-corpuscles, rapidly tend to become converted into purulent exudates after any major operative intervention.

An experience extending over many years convinces me that when puncture performed with the usual antiseptic precautions is followed by suppuration or putrefaction, it is not because large masses of infective micro-organisms have been introduced into the pleura, for it is found, on the one hand, that puncture of the abdominal cavity in ascites, even under the most unfavorable conditions and when repeatedly performed, is not always followed by suppuration or peritonitis, and, on the other hand, it must be remembered that any minute quantities of micro-organisms that may adhere to the instrument are wiped off in the external tissue at the time of puncture.

Hence infection by means of the syringe or trocar would have to show itself in an inflammation of the puncture-wound, which we know is a comparatively rare event, and even when it occurs may have been caused by subsequent infection of the open channel.

While I cannot, therefore, subscribe to the view that the infection accompanying puncture is always the cause of a diminution in the exudate, I do not go quite so far as to exclude altogether the possibility of an injurious effect following mechanical intervention under certain conditions. I do not deny that puncture followed by aspiration and sudden evacuation of the pleura may act as a distinct mechanical irritant, especially when there is much inflammation at the time of the operative intervention, and too much of the fluid is withdrawn, or a large trocar is moved about in various directions. It is also possible that a very intense mechanical irritation of this kind may be the direct cause of an intensified tissue activity, which we call suppuration, whenever there is a predisposition to increased reaction.

When *putrefaction* takes place, the presence of carriers of infection is always to be presumed. They enter through the lymph-channels of the pulmonary pleura or they are carried in by migratory cells entering the pleura from the lung; but even in putrid exudates the bacterial etiology is not always distinctly demonstrable, as micro-organisms are not always found.

A spontaneous decomposition of an albuminous fluid, in our opinion, is not beyond the realm of possibility, since the preservation of a definite composition in fluids and secretions of cavities depends on the preserving influence derived from a normal functionating tissue in the wall of the hollow organ. At least it must be admitted that the conditions which obtain when sterilization is effected by exposure to very high temperatures are not identical with conditions of asepsis in the organism.

The conclusion arrived at is, therefore, that bacteriologic investigation has failed to explain the most important questions in the etiology of pleurisy. The presence of specific pyogenic micro-organisms, streptococci and staphylococci of various forms, is not necessary for the conversion of a serous into a purulent exudate, and the necessary conditions for the formation of pus are as much hidden from our knowledge to-day as are the various causes of inflammation of the pleura.

The many hypotheses offered for the explanation of these difficulties do not suffice to bridge the chasm existing between clinical observation and the very variable results of bacteriologic examination. The explanation advanced by a number of authors, that the production of a purulent process depends on the number and virulence of the micro-organism present, fails to explain; it is merely the old saying in a new garb, that the reaction depends on the quantitative and qualitative nature of the irritant. But even this view of the action of micro-organisms is not borne out by the results so far obtained, since inoculation with micro-organisms present in smaller numbers, and therefore presumably more virulent, is not by any means always followed by any more dangerous manifestations than inoculation by other micro-organisms, the virulence of which may be doubted from the fact that they are present in large numbers in comparatively small exudates.

[**Synopsis of Bacteriology of Pleuritis.**—Setting aside the influence of cold as a predisposing agent, our knowledge of the etiology of pleurisy may be summed up as follows:

The *serofibrinous*, or so-called spontaneous or idiopathic pleurisy, in view of recent investigations, appears to be due to an irritation brought about by the products of micro-organisms. Most pleurisies can be accounted for by either one of three micro-organisms: tubercle bacillus, Diplococcus lanceolatus, or Streptococcus pyogenes.

Our knowledge of the etiology of pleurisy has been greatly broadened by the work of French investigators, especially that of Netter. His article in the fourth volume of the "Traité de Médecine" gives a very complete consideration of this subject.

1. *Pleurisy due to the tubercle bacillus:* In support of this type of pleurisy we have first certain clinical and pathologic observations, as: (*a*) tendency to pleuritis in tuberculous subjects; (*b*) association of tuberculosis with the serofibrinous type of pleuritis; (*c*) pleurisy coming on abruptly in healthy persons has been shown by autopsy or by subsequent history to be tuberculous; (*d*) those who recover from this type of pleurisy frequently develop pulmonary tuberculosis later. Of interest in connection with these facts are the observa-

tions of Henry J. Bowditch, who found that 32 out of 90 cases of acute pleuritis under observation from 1849 to 1879 died with lesions of pulmonary tuberculosis. Of 131 cases of pleuritis of all types, examined postmortem, Osler found 32 to be definitely tuberculous.

Bacteriologic examinations, either by cover-clip preparations or by cultures, in tuberculous pleurisy are practically always negative, as shown by the work of Prudden, Grawitz, Ehrlich, Ferdinand, Le Damany, Eichhorst, Netter, Aschoff, Levy and Klemperer. This fact has led many observers to consider negative bacteriologic results as strong presumptive evidence of the tuberculous origin of these cases.

Of recent years inoculation experiments have placed the theory of the tuberculous origin of pleurisy on a firm foundation. Pansini inoculated pleural exudate from 15 cases; in 6 cases tuberculosis developed in the inoculated animal. Netter obtained positive results in 8 of 16 cases known to be tuberculous, and 10 of 25 cases in which pleuritis was supposed to be due to exposure to cold. Fourteen cases which were explained on other grounds were all negative. As a result of this work, Netter believes that 40% of all primary exudates are tuberculous, and that in serofibrinous pleurisy the tubercle bacillus is the most common agent.

In another group of cases reported by Netter,* in which purulent exudates from 109 cases were examined, 12 were found to be tuberculous. Aschoff † examined 220 serous exudates, 193 of which were negative microscopically and by cultures. In 37 of these cases animals were inoculated, tuberculosis resulting in 68%. Of these 37, 12 were so-called idiopathic pleurisy, tuberculosis resulting in 75%.

Equally striking are the results obtained by Eichhorst, who was able to obtain a considerably larger proportion of positive results by inoculating larger amounts of exudate. He used at least 15 c.c. of exudate, and was able to demonstrate tuberculosis in more than 62% of his cases.

Le Damany, by using even larger amounts of exudate, in doses from 10 to 50 c.c., sometimes giving an animal as much as 300 c.c. of exudate, was able to demonstrate the tuberculous character of all but 8 of 55 primary pleurisies. In 4 of the remaining cases, however, concomitant lesions indicated a tuberculous etiology, thus making all but 4 cases of tuberculous origin. He found cultures universally negative. As a result, he divides all pleurisies into two classes: primary or tuberculous, and secondary or non-tuberculous.

Levy and Klemperer state that serofibrinous as well as purulent pleuritis may be due to a tubercle bacillus.

2. *Pleurisies due to Diplococcus lanceolatus:* Under this grouping we have most of the so-called "primary empyemas" of children as well as the serofibrinous or purulent pleuritis secondary to pneumo-

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* *Rev. Gén. de Clin. et de Thérap.*, 1890, No. 21, p. 341.
† *Zeit. f. klin. Med.*, 1896, xxix, p. 440.

nia. This fact has been shown by the observations of Netter, who found the pneumococcus in 32 of 109 cases of purulent pleuritis. Pansini, Prudden, Levy and Klemperer, and Aschoff have had similar results. Wright and Stokes* report 16 cases of fibrinous pleuritis, and 6 of empyemas; 13 of the former and 4 of the latter accompanying pneumonia, in all of which Diplococcus lanceolatus was found. Pearce,† in a study of 121 fatal cases of lobar pneumonia, found acute pleuritis in 49; Diplococcus lanceolatus being present in all.

The prognosis of pneumococcus pleurisy is much more favorable than that due to other micro-organisms. Vierordt, however, has recently reported four cases of pneumococcic empyema with a fatal termination; in two of these cases peritonitis due to the same organism was present.

3. *Pleuritis due to streptococcus:* Occurs chiefly in empyemas in adults, and is usually secondary to inflammations of contiguous structures, as pneumonia, bronchopneumonia, gangrene, abscess of lung, wounds or inflammations of chest-wall, or structures of the neck; occasionally by extension from the peritoneum, or by general infection through the blood.

Cases of streptococcus pleuritis have been reported by numerous investigators, among whom may be mentioned Pansini, Netter (who found it present in 51 of 159 purulent exudates), Levy and Klemperer, and Aschoff. In children it is frequently associated with diphtheria, scarlet fever, measles, and occasionally with whooping-cough. Pearce‡ found it to be the common organism in the pleuritis of scarlet fever. Councilman, Mallory, and Pearce,§ in a study of 220 fatal cases of diphtheria, found that the streptococcus, either alone or associated with other organisms, was a common factor in the pleuritis of diphtheria and scarlet fever.

Many other organisms are occasionally found in pleuritis; thus, Staphylococcus pyogenes aureus is frequently found in pleuritis secondary to suppurations of other parts of the body. Friedländer's bacillus, colon bacillus, diphtheria bacillus, Micrococcus tetragenus, and typhoid bacillus are also occasionally observed. In pleuritis due to the latter micro-organism cases have been reported by Charrin and Rogers, and by Sahli and Frenet. Labiche‖ states that this form of pleurisy may be independent of diseases of the lung, and may be either serous or hemorrhagic, though most often purulent. Achard** has reported two cases in which the typhoid bacillus was obtained in pure culture. A doubtful case of gonococcus pleurisy is reported by Jicinsky. Tubercle bacilli were absent from the exudate, which contained numerous diplococci thought to be gonococci. No cultures are reported. There was a history of antecedent gonorrhea which appears to have been the chief support of the diagnosis. Welcke found a parasite which resembled Cercomonas intestinalis. It was thread-like, with a spindle-shaped enlargement at one end. All the parasites were active. No operation was performed and the parasites disappeared from the pleural cavity after a time. The parasites did not grow on culture-media ; injected into the pleural cavity of a rabbit they produced only pus containing staphylococci.* Hamilton investigated the bacteriology in 23 out of a series of 30 cases of empyema. Streptococcus, Staphylococcus, and Diplococcus pneumoniæ occurred each with about equal frequency, either alone or in combination with one another and with Bacillus tuberculosis and Bacillus coli communis. Bacillus ramosus, Bacillus pyocyaneus, Bacillus subtilis, and Bacillus tuberculosis were found once each.† May and Gebhard‡ report a case of pneumothorax due to the presence of a gas-forming bacillus. A stab wound in the cardiac region healed, but was followed by the development of a hemorrhagic effusion, with diplococci, in the left pleural cavity. Later pneumothorax appeared and the fluid became purulent. The pleural cavity contained bacilli which produced carbon dioxid, hydrogen, and nitrogen. Cultures of Bacillus coli communis and Staphylococcus were also obtained. In the putrid effusions occasionally met with, organisms of putrefaction, especially proteus, are found.

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* *Boston Med. and Surg. Jour.*, 1895, cxxxii, p. 271.

† *Boston Med. and Surg. Jour.*, Dec., 1895.

‡ *Reports of Boston City Hospital*, 1899.

§ *Journal of Boston Society of Medical Sciences*, vol. v, No. 5, 1900.

‖ *Gaz. hebdom.*, 1899, No. 17.

** *Le Semaine Medicale*, Oct. 9, 1898.

The exudates of pleuritis associated with articular rheumatism and carcinoma are generally sterile.

To sum up, pleuritis is usually due to one of the three micro-organisms above named. There is, however, no uniform bacteriology, for other micro-organisms may occasionally produce the lesion, and mixed infections are not uncommon. The pyogenic cocci as well as the tubercle bacillus may produce either serofibrinous or purulent exudate. The so-called idiopathic pleurisy is usually due to the tubercle bacillus; that secondary to lobar pneumonia is in most cases due to Diplococcus lanceolatus, that secondary to other inflammatory conditions of the lung, or of neighboring structures, is usually due to the streptococcus, occasionally to Staphylococcus aureus or other organisms.—Ed.]

VI. THE TISSUE CHANGES.

The changes occurring in serous membranes, especially in the pleura, in the various stages of inflammation have been carefully studied both clinically and experimentally. At the beginning of inflammation circumscribed areas in the pleura appear opaque, are rough to the touch, and are traversed by numerous branching vessels. The surface of the membrane is covered by fibrin deposits of various shapes, some linear, some in the form of a network, others ribbon-like or nodular; in the intervals between these fibrin coagula, and on the denuded serous membrane after their removal, ecchymoses of varying extent are found. The endothelium in these cases is converted by a form of *coagulation necrosis* into the superficial product of the inflammation known as the **exudate.** The exudate is usually more pronounced on the costal than on the pulmonary pleura, where the inflammatory process, as a rule, shows itself only in the region of the interlobular fissures. As a result of this form of coagulation necrosis, which is accompanied by the formation of a layer of fibrin, **adhesions** of varying density and extent are formed in the regions of the apex and root of the lung. Where the excursions of the organ are more extensive, as at the base and margins of the lungs, there are no adhesions; the gelatinous and more elastic exudate is spread out over larger portions of the organ and drawn out into ribbon-like bands by the respiratory movements.

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* *Münchener med. Wochenschrift*, August 23, 1898.

† *Montreal Medical Journal*, October, 1900.

‡ *Deutches Arch. für klin. Med.*, vol. LXIII.

The reason, for instance, that in many cases of phthisis the pleural adhesions are extensive above and ribbon-shaped below is either that the respiratory displacement varies in the two regions or that the inflammatory process over the lower segments of the lung is less intense.

When there is a moderate amount of exudate, the pleura is much more succulent than usual, and even in the subserous tissue there is an increased turgescence and accumulation of lymph and lymph corpuscles. After the acute inflammatory stage an active new-formation of blood-vessels in the pleura and in the exudate takes place which is followed by absorption of the fluid portions of the exudate, so that the two layers of the pleura become more approximated. Later the solid deposits break down, the adherent lamella undergoes fatty degeneration, whereupon the products are taken up by the newly formed blood-vessels and by migratory cells, and thus re-enter the vascular system. The process ultimately ends in the complete disappearance of the exudate, or the pleura remains entirely covered by a layer of connective tissue which may be of considerable thickness; or a thin band of connective tissue resembling loose subcutaneous cellular tissue may form between the two layers of the pleura.

According to Ackermann,[2] the large newly formed vessels within the adhesions of the pleura serve to carry off the blood from the altered pulmonary tissue and thus lay the foundation for a collateral circulation. Schlenker's [2] theory, that fibrinous pleurisy is always followed by pleural adhesions because an exudate cannot break down and become absorbed on an endothelial surface, does not appear tenable, since it is quite possible for cells and liquefied portions of tissue to be carried off by the numerous vessels which are formed in the pleura, and both endothelial and migratory cells possess the property of taking up fatty or otherwise degenerated corpuscular elements. But as soon as the inflammatory product has been removed and the abnormal stimulus for the activity of the newly formed vessels disappears, the vessels necessarily atrophy from want of work, and are converted into connective tissue, or may even be completely absorbed by the migratory cells. Hence the former sites of extensive adhesions and other tissue changes are often indicated only by an opacity or slight thickening of the pleural tissue. The pulmonary layer of the pleura appears to be chiefly concerned in these resorptive processes.

Another series of tissue changes takes place when a **fluid** is formed in addition to a dense deposit on the surface of the pleura, often consisting of several distinct layers. There is in such cases an abundant accumulation of inflammatory products; the fluid may be light yellow or yellowish-green, frequently opalescent, and sometimes reddened by the admixture of blood; it may be clear or turbid, either from the presence of coarse shreds of fibrin or cells, or because it contains minute blood-clots.

The pigment of the exudate is probably closely related to lutein, which is regarded by Krukenberg [1] as the pigmentary element in blood-serum. Bile-pigment, according to my own observation, is found only in jaundice. In an exudate which became blue on exposure to the air, P. Guttmann found indican, from which indigo is derived. A similar observation was made by C. Gerhardt.[1]

The *specific gravity* varies between 1005 and 1035; the *amount* ranges all the way from a few cubic centimeters to many liters, the average amount being from 1 to 2.5 liters. The fluid when left to itself slowly coagulates outside the body, but the coagulation may be accelerated by heating. On standing, a heavy sediment consisting of degenerated red blood-cells and pus-corpuscles in various stages of fatty degeneration is precipitated; occasionally well-preserved red corpuscles are also found. As a rule, the specific gravity is greater in proportion to the abundance of foreign matters, such as blood, pus-corpuscles, and fibrin shreds, present in the fluid; since the more morphologic elements are present, the more concentrated will be the lymph that is formed. On chemical examination the fluid is found to resemble blood-serum in its constituents, containing urea, cholesterin (not constant), sugar, and peptone; glycogen is not present. Oxygen and nitrogen, according to A. Ewald,[9] are not found in pleural effusions; on the other hand, they contain large quantities of carbonic acid.

A transudate (due to stasis) is said to contain less chlorids than an exudate, although the amount of salts is the same in both; the difference in the quantity of chlorids is, however, very slight. The exudate may contain as high as 7% albumin; if more than 3% of albumin is present, the condition, according to some authors, is always due to inflammation. The formulæ given by Reuss and Runeberg for the relation between the percentage of albumin and the specific gravity do not appear to be of any great practical value.

The greater the mechanical obstruction to the removal of lymph offered by the inflammation, the more limited will be the passage into the blood of lymph, which is secreted in abnormally large masses by the irritated tissue. Accordingly, it makes its way into the reserve spaces of the tissue, the interstitial clefts, and into the large pleural cavity, which is the most convenient lymph-space at hand, and this materially modifies the appearance of the tissue changes.

As the solid deposits are separated into distinct layers, cavities are formed in which the fluid exudate undergoes various alterations due to absorption, the entrance of cells, and to additional exudative processes. In this way isolated or communicating cavities are formed which are known as **sacculated** or **multilocular pleurisy.** Judging from the results of exploratory puncture it does not seem probable that the fluid, in the absence of partitions of this kind, always seeks the lowest level, since the effect of gravity may be materially interfered with by aspiration when the function of the lungs, the thoracic muscles, and the diaphragm are fairly well preserved. In other words, the fluid in the pleural cavity is always found at a *higher level* than gravity would seem to indicate, just as in a capillary tube whose walls are moistened by the contained fluid.

It follows, therefore, that the reason why the greater portion of the exudate is found in the lower portions of the pleural sac is not exclusively because the exudate tends to sink with the effect of gravity, but also because in these portions the *resistance to the fluid exudation is much less than elsewhere.* As the tension of the lung is greatest in this portion, it offers more resistance to the contraction of the muscles if the tonic innervation is not relaxed at the proper moment of inspiration, as under normal conditions. Moreover, a foreign medium prevents expansion in this direction even when the so-called secretory pressure is comparatively low, because the abnormal stimulus in the inflamed pulmonary pleura acts in the same direction and materially reinforces the tonic expiratory stimulus.

It must also be remembered that the distention of the lower portions of the lungs demands absolute precision in the mechanism of respiration and a specially vigorous action of the muscles of the thorax and of the diaphragm, because in forcing the air into the lowest portions of the lung its natural tendency upward must be overcome. This is probably the chief explanation why a solid exudate is almost always found over the apices and a fluid exudate in the lower portions of the sac, and it also explains why the adhesions at the apices are, as a rule, when the other conditions of the lung are normal, more dense than in the lower portions.

Absorption.—The power of the pleura to absorb fluid exudates varies according to what has already been said. In general, absorption is more difficult in proportion to the firmness and extent of the deposits on both layers of the pleura from the beginning, since they indicate a high grade of irritation in the layers of the pleura from which the exudate is secreted; and the abundance or the number of the precipitated elements from the blood is an index of the strength of the irritant or the extent of internal tissue activity. The greater the obstacle to the external work,—the work required for the transportation of materials into and away from the blood-vessels,—and the greater therefore the internal activity that is concerned in inflammation, the greater will be the difficulties in the way of re-establishment of normal conditions. When this obstacle to transportation reaches a certain degree, the exudate either cannot be absorbed at all, or if it is removed by artificial means, it reaccumulates in a short time, since the means are entirely wanting for the removal of the lymph which is formed in constantly increasing amount by the irritated tissues. The most favorable event is the precipitation of dense masses of fibrin which afterward organize and form connective-tissue bands of great width and density.

Complete **obliteration** or synechia of the pleural sac rarely occurs in serous pleurisy with the present methods of treatment; **calcification** and **ossification** occur only after a badly healed suppurative pleurisy.

To test the power possessed by the pleura of absorbing corpuscular elements Fleiner [1] injected large masses of defibrinated blood or finely granulated India ink into the pleural cavity of animals, with careful precautions against the entrance of air, and killed the animals after ten to fifteen minutes. He found that within a very short time corpuscular elements were taken up, carried off in the lymph-channels of the pleura, and deposited in the lymph-glands of various regions. The absorption evidently takes place in the costal and mediastinal pleuræ, to the exclusion of the pulmonary pleura. The pigment granules were absorbed by the costal pleura, assisted in part by the action of the respiratory muscles, and carried into the intercostal, but never into the bronchial, lymph-glands. The particles coming from the mediastinal pleura were deposited in the peritracheal lymph-glands above the bifurcation. Since impaction of the lymph-glands offers an obstacle to absorption, Fleiner believes that a failure of pleuritic or pneumonic exudates to undergo absorption is more often due to changes in lymph-glands than has hitherto been supposed.

If the inflammatory bands undergo retraction, or, to speak more correctly, if the lung, on account of the impaired function of its tissue or the impairment of the entire respiratory mechanism, is unable to expand, the form of the **thoracic cavity** undergoes marked variations; it becomes contracted and distorted in all its diameters. Owing to the atrophy of the thoracic muscles and the formation of contractures from the absence of antagonistic muscles and the ossification of ribs and joints, the welfare of neighboring organs may be interfered with in various ways, and often with very serious consequences.

The *heart*, the *liver*, the *mediastinum*, become displaced or compressed and their functions disturbed by interruption of the usual nervous influences; these abnormal stimuli, in turn, set up other inflammatory processes in the immediate vicinity and, by interfering with compensatory activity, prevent the organism from regaining its equilibrium.

One of the most important varieties of the ordinary fluid product of exudative pleurisy is the **hemorrhagic** form, which may present every shade of red or brown discoloration. Brownish-red as a rule, it sometimes appears bright red; in endothelial cancer of the pleura it is said to be black, like the blood in venesection (A. Fraenkel[3]). The number of red blood-corpuscles is small if the upper layers only are aspirated; in the lower ones it is considerably greater. The red cells are often perfectly preserved; more marked alterations are usually seen when the exudate contains large numbers of pus-corpuscles; rarely hematin crystals are also found.

In one case of sacculated pleurisy of ten years' standing, with a brownish-red exudate, in a woman affected with tuberculosis, I found, in addition to cellular detritus, blood pigment, etc., a number of the most beautiful cholesterin crystals. At the autopsy the crystals were found accumulated in large numbers in the brown, villous deposits, where they formed regular hyaline bodies (drusen).

A hemorrhagic exudate is the expression of increased activity of the vessel-walls accompanying distention of the pleural blood-vessels, —be it an active, acute (congestive) hyperemia, due to inflammation, or a passive hyperemia due to interference with the flow of blood,— and indicates a special disturbance in the tissues of the vessel-wall which we designate as an abnormal permeability, the walls of the vessels, even when the tension is subnormal, being unable to retain their contents. This form of diapedesis always indicates local or general, relative or absolute weakness.

Accordingly, hemorrhagic exudates are found, on the one hand, in individuals of good constitution, when they indicate a maximum increase in local tissue activity following an abnormally strong irritant; and, on the other hand, in marasmus, in alcoholics, in senile and decrepit individuals, in the presence of malignant tumors, in constitutional disease, in scurvy, etc., when they indicate a profound nutritive disturbance. The nature of the exudate alone, therefore, indicates merely a temporary (relative or absolute) inability of the vessel-walls to retain the red blood-corpuscles. It is only when this insufficiency becomes permanent that we are justified in assuming a true permeability, the sign of absolute debility and cachexia. [A case of hemorrhagic effusion into the pleuræ and into the peritoneum, reported by Cheeseman and Ely,* is to be regarded as a case of non-malignant, non-tubercular, primary hemorrhagic effusion. The pleural cavity was aspirated from twenty to thirty times within a year, and bloody fluid was obtained on each occasion. The patient had a fibroid of the uterus. The effusion was finally cured by the injection of Thiersch's fluid and iodin. Several weeks later an accumulation of hemorrhagic fluid began to form in the abdominal cavity and persisted for four years, finally ceasing with the menopause. Fernet * describes the occurrence of a hemorrhagic pleural effusion in an alcoholic subject.—Ed.]

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* *American Journal of the Medical Sciences*, August, 1899.

The basis for the tissue alterations in inflammatory hemorrhagic disease of the pleura is precisely the same as in hemorrhagic pachymeningitis and similar processes; one of the chief causes for the admixture of blood in the exudate, after the abundance of newly formed vessels, is the relative thinness of the vessel-walls.

Since, therefore, a hemorrhagic exudate is often no more than an index of the great number of vessels in the pleural deposits, one is not justified in regarding it as directly pathognomonic of tuberculous pleurisy, although as a matter of clinical experience it frequently occurs in that form; in all but the most rapidly fatal cases of tuberculous pleurisy the exudate to the end remains serous. Tubercle bacilli have occasionally been found in serous exudates. Personally, I have never been able to find them in a closed pleural cavity.

It is difficult in all cases to draw a sharp dividing-line between serous and purulent exudates, just as between hydrothorax and a serous exudate. In general, it may be assumed that a primary purulent effusion, even after pneumonia, is relatively rare; a serous or hemorrhagic exudate may, however, become converted into a purulent exudate within a very short time.

Primary purulent effusions are usually encapsulated and of small extent; they are, as has been mentioned, most frequently found as so-called metapneumonic exudates in the wake of croupous pneumonia or in pyemic processes. They are more common in children than in adults, at least there is no doubt that a serous exudate in children is converted into a purulent exudate in a much shorter time.

Hagenbach-Burckhardt[7] makes the somewhat remarkable assertion that in 26 cases of purulent exudate in children, 25 occurred immediately after croupous pneumonia and only 1 after scarlatina, and that in a number of these cases suppurative processes were found in other organs which were to be regarded as due to metastasis or secondary infection in a debilitated organism.

To judge from our own observations, true croupous pneumonia is rarely followed by empyema and pleurisy in children. The pleurisy is usually acute and develops without any assignable cause as a sequel of inflammatory processes and infectious diseases, or after those forms of bronchitis which rapidly lead to the formation of bronchopneumonic foci. We have also observed pleurisy following whooping-cough, but in these cases an element of doubt remained whether the severe paroxysms were not the result of a latent central focus in the lung.

A single exploratory puncture does not suffice to determine the nature of an exudate, and it must not be forgotten that the different layers of an exudate may contain different materials. Since the heaviest portions tend to sink to the bottom, it is often found that a serous exudate containing abundant round cells consists of two or even three layers, the highest of which is clear and transparent, and of lower specific gravity than the deeper ones, which usually contain all the corpuscular elements present. Thus, if the upper layer is first aspirated, and after a few days the puncture is repeated in the lower layer, one would be apt to infer that a serous exudate had become converted into a purulent one, whereas, in reality, one simply has to deal with a serous exudate containing many cellular elements. A purulent exudate may, as has been mentioned, present the widest variations of color and density. It is almost never as thick as the pus from an old abscess cavity; usually it contains small quantities of blood. In metapneumonic exudates it is more mucopurulent, the color variations ranging all the way from yellowish-white and yellowish-green to brownish-yellow and brownish-red. The specific gravity varies widely. The exudate contains numerous cells of varying size and form in all stages of degeneration; cells containing several small nuclei or a single vesicular nucleus filling the entire cell-body are not uncommon. Usually these morphologic elements are derivatives of the white blood-corpuscles; sometimes, however, they show the characters of pleural endothelium. All kinds of bacteria are found by appropriate staining.

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* *Soc. Med. Par.*, June 22, 1900.

The peculiar fatty character of the empyema fluid in tuberculous subjects, which often resembles a fatty emulsion, and is due to extensive degeneration of the many cellular elements which it contains, has led to the adoption of a special term—*empyème graisseux* (Bouveret [1]).

Purulent exudates may undergo spontaneous absorption, but this event is not as common as *rupture* into a bronchus or some other organ. Occasionally when empyema is left to itself, *encapsulation, inspissation,* and *caseation,* or conversion into dense plates may take place. If encapsulation occurs, the various cavities may at the same time contain serous and purulent fluid.

A **putrid exudate** is usually greenish-yellow in color; more rarely yellowish-brown or chocolate brown. It emits a cadaverous odor; macroscopically numerous plugs consisting of detritus and micro-organisms are found, and on microscopic examination disintegrated cells of various forms, hematin crystals, needles of fatty acid and free fat, leucin, tyrosin, and cholesterin, as well as micro-organisms, are seen. In rare cases an apparently normal serous exudate may be fetid in character.

The question whether putrefaction of the pleural effusion can occur primarily—that is to say, without the existence of a pulmonary or thoracic fistula—should, I believe, be answered in the affirmative. In the first place, an exudate always contains the germs of disintegration, and when it is in a measure left to itself, and the vital conditions in the surrounding membranes have suffered a certain alteration, the fluid may readily undergo decomposition without coming into direct contact with the external atmosphere, and, in the second place, the possibility of the entrance of the specific micro-organism of decomposition can never be excluded in a cavity the walls of which are not hermetically sealed, but keep up a constant metabolism with surrounding structures by means of numerous clefts or tears and other channels.

In a word, micro-organisms and other exciting causes of putrefaction may reach the exudate by the continuity of the tissues whenever the vital powers of the pleura are diminished, and additional chances for putrefaction to take place are afforded by the presence of putrid processes in neighboring portions of the lungs, not to mention disease of the other adjoining organs. In destructive processes, such as caries of the vertebræ, esophageal or gastric tumors, carcinoma, gangrenous foci in the lungs, pneumothorax, the rupture of tuberculous cavities, it is impossible to prevent the ultimate putrefaction of the pleural contents.

The form described by Fräntzel as *pleuritis acutissima* appears to possess a special tendency to putrefaction, probably because under the influence of the greatest possible inflammatory stimulus present in that variety the vital power of the tissue is rapidly destroyed. The same conditions which destroy the power of the tissue to perform the necessary work for preserving its integrity obviously suffice to bring about the disintegration of the tissue itself and of its contents.

It has already been stated that serous and purulent fluids may exist side by side in the various cavities of a multilocular pleurisy, and we may add that serous, putrid, and hemorrhagic exudates may also be present at the same time. Even a purely serous fluid may emit an extremely fetid odor.

This odor in a serous exudate is not always the product of decomposition or putrefaction of the exudate itself, but may be inherent in the fluid; its origin may be, for instance, a gangrenous focus outside of the pleura, within the lung, or in one of the neighboring organs. Similar conditions occur, as we know, in the peritoneum, where any variety of accumulated fluid, especially thin pus, is very apt to develop a fecal odor even in the absence of intestinal perforation.

Purulent and putrid effusions not infrequently lead to extensive **necrotic processes,** erosions, ulcers, perforations, and abscesses due to congestion and stasis. Necrosis of the pulmonary pleura is usually followed by rupture into the bronchi, while necrosis of the costal pleura most frequently leads to undermining of the intercostal muscles, bulging of the skin, and the formation of an abscess rupturing on the surface of the body—*empyema necessitatis.* Direct rupture through the diaphragm is extremely rare. Extension of the inflammation by contiguity to the peritoneal cavity is much more common. On the whole, involvement of the peritoneum in abscess of the pleura appears to be relatively less common than the formation of a pleural exudate in peritoneal diseases, especially if evacuation of the fluid, either external or internal, is delayed. Pyemic processes not infrequently follow an untreated or incorrectly treated pleurisy.

If so-called pyogenic membranes develop on both layers of the pleura, a constant renewal of the exudate takes place, and even after the fluid has been withdrawn either by puncture or through some internal channel, recovery is delayed as long as the tendency to secretion continues. Such patients usually die of exhaustion from the constant formation of pus, or of fatty heart, or amyloid degeneration.

It may be mentioned here that erosion, necrosis, and finally rupture are not directly produced by the action of the exudate—either the pressure or any peculiar chemical corrosive quality of the fluid; they are the result of the exudative irritant, which, as is shown by the process of healing, resides partly in the pleura but chiefly in the exudate itself and produces a constantly increasing tendency to pus formation; that is to say, the demand on the local external activity of the tissues is so great that the local, internal work necessary for the maintenance of the integrity of the tissue eventually suffers, or, in other words, necrosis and disintegration must take place unless the irritant is removed.

It may not be amiss also to point out once more that the pyogenic membrane is not the cause of pus formation. On the contrary, the formation of a true pyogenic membrane is the result of insufficient expansion of the lung, and is the characteristic expression of a maximal irritation in the pleura—an effort, so to speak, to fill, by uniting the layers of the pleura or forming a plastic exudate, an abnormal space which would not exist if the lung expanded sufficiently and the function of the respiratory mechanism were perfect. The costal pleura and the thoracic wall are unable to perform the external (mechanical) work of aspiration if they are occupied in producing abnormally large masses of inflammatory products; that is to say, are doing chiefly plastic work. The production of a pyogenic membrane or of large plastic deposits merely shows in many cases that the therapeutic measures are not sufficiently reinforcing the natural regenerative process, which demands, above all, the expansion of the primarily infected lung and the evacuation of the exudate, or that the treatment has come too late and therefore could not contribute to the cure of the condition.

In concluding the discussion on the causes of the production of the different varieties of exudate, we are forced to admit that several points still demand careful study. Conversion of the exudate into pus is probably due in every case to the appearance of specific infectious agents or chemical substances, the source of which we are at present unable to determine, since the same variety of bacteria and identical external influences are observed to produce different varieties of exudate, and in many cases it is difficult to believe that this difference is due solely to increased virulence of the respective bacteria or unusual magnitude of the irritant agent. Again, disposition, or, in other words, differences in reaction, play an important rôle, and it is much to be desired that more attention be paid to this factor in explaining mixed infections; that is, cases in which different varieties of micro-organisms are found in the same exudate at the same time.

In all extensive pleurisies, particularly in the acute purulent forms, there are found, in addition to the alterations within the thorax, changes in the thoracic walls, edema of the subpleural tissue, inflammation of the intercostal muscles, and a more or less extensive and brawny edema of the skin, especially in the lateral walls of the thorax, the degree of which is best determined by pinching up a fold of skin at corresponding points on the chest-wall.

Having in the foregoing discussed the changes and ultimate results dependent upon the quality of the exudate, it remains to say a few words concerning the equally important phenomena due to the quantity of the fluid.

VII. MECHANICAL EFFECT OF THE EXUDATE.

If we assume the air-contents (vital capacity plus residual air) of the lungs in a vigorous adult, not affected with emphysema, to be 4500 to 5500 or 6000 c.c., we shall get an approximate idea of the degree of compression of the lung and the diminution in the respiratory surface which would have to result, even from a moderate accumulation of fluid, if it were not for the fact that the neighboring organs are displaced and thus tend to maintain an adequate respiratory surface.

The consequences of the so-called **pressure,** however, also manifest themselves in a diminution of the volume of the lung due to atelectatic processes. The organ may by retraction gradually become airless (atelectatic), and, if the effusion is very great, the direct compression may convert the lung into a dense, dark-brown, compact body which usually occupies the region of the former hilum between the thoracic wall and the vertebral column, but may also be displaced upward or directly backward by the action of adhesions or other abnormal conditions. Under favorable circumstances it may, after the effusion has become absorbed, completely regain its distensibility. In addition, other changes result, due only in part to pressure or the action of gravity; dislocation of the mediastinum and of the heart, kinking of the large vessels, flattening and depression of the diaphragm, and dilatation of the thoracic cavity.

The mechanism of dislocation by an exudate deserves especial consideration, as the difficulties in the way of a satisfactory explanation have not been entirely removed by the great number of experiments and clinical investigations. Of late years the dynamic (biologic) conditions, the tone of the tissues, which is the basis of the vital manifestations, has been somewhat neglected in favor of what I should like to call the *static conditions*.

The usual explanation of these processes is as follows:

The expansion of the lung, as is well known, depends on three factors: (1) The atmospheric pressure against its inner surface, by virtue of its free communication with the atmosphere through the bronchi; (2) the pressure acting on the outer surface (pulmonary pleura) of the lung; and (3) the intrinsic elasticity of the organ. As, however, the external pressure does not exist under normal conditions, because the bony and muscular walls of the thorax, which is air-tight and contains the lungs, support the weight of the atmosphere, only two factors—the *internal pressure* and the *elasticity*, which work in opposite directions—come into consideration. While the former tends to expand the organ, the effect of the latter is to contract the lung concentrically and to reduce it to the volume which it assumes when it is taken out of the thorax.

It follows, therefore, as a consequence of the air-tight conditions within the thorax, that the lung is forced by the enormous excess of pressure on its internal surface to follow the dilatation of the thoracic walls to their extreme limit, and it is only when this internal pressure is to a certain extent neutralized by the corresponding external pressure which occurs when the thoracic walls collapse in expiration that the action of the elasticity of the lung can bring about a diminution in the size of the organ by active contraction.

This neutralization obviously cannot take place if there is a positive pressure in the pleural cavity, or, rather, if the presence of a foreign medium permits of a pleural cavity being formed, or if an increase in the intra-abdominal pressure forces the diaphragm against the thoracic cavity, so that the pressure equilibrium can be re-established only by an alteration in the tension and position of this dividing wall at the expense of the volume of the lung.

In the same way a diminution in the thoracic space occurs when the action of the thoracic muscles, which work in the same direction as the intrabronchial pressure, is impaired by the interposition of a foreign medium. The greater the pressure exerted on the pleura, the greater the compensatory internal pressure and the greater the demand on the elasticity of the lung or on the active forces—the intercostal muscles and the diaphragm, which are then required to counteract the spontaneous contraction of the lung and thus to achieve the same expansion as before.

For the full effect of the elasticity of the lung—that is to say, the retraction of the lung as seen in the organ when it is removed from the body—to make itself felt, the external pressure must be equal to the pressure of the atmosphere, or, in other words, to the internal pressure, hence a true compression of the lung can take place only when this point has been reached; that is to say, a condition when the walls are approximated not by the effect of active elasticity, but by an excessive external pressure, and when the bronchial walls, which normally are held apart by their cartilaginous rings, are so compressed that all the air escapes, thus leading to the well-known picture of compression atelectasis with occlusion of the alveoli and blood-vessels.

On careful consideration of individual cases, however, it is found that this, at first sight plausible and simple explanation does not completely suffice to elucidate the mechanism of compression of the lung, because the physical conditions are too complicated to be explained by the mere assumption of some alteration in the external and internal air-pressure.

It has been clearly shown by experiment that it is not always possible, even when employing considerable force, to introduce a foreign medium into the pleural sac when the lung is intact, and we find from clinical experience that the formation of atelectasis bears no proportion to the degree of pressure exercised by the exudate. Leyden,[5] Quincke,[5] Homolle,[5] and Schreiber[5] found by actual measurement that even in the presence of very large exudates the increase in the intrapleural pressure was scarcely equal to one-twentieth of an atmosphere; in many cases it even fell below this limit.

How, then, is it to be explained that complete compression of the entire lung often occurs when there is only a moderate effusion? and how is it that often only one portion of the lung—the lower lobe—becomes compressed and atelectatic, the other portions appearing to be, if anything, expanded?

The answer to these questions is readily found if we consider a factor which has received too little attention in the past—namely, the effect of **tonus** on the mechanism of respiration in pleural effusions.

Since, as has been already explained, the lung, on the one hand, follows the inspiratory pull of the diaphragm and other respiratory muscles as the thorax expands, and the diaphragm, on the other hand, as it relaxes follows the pull of the retracting lung, a moderately large pleural effusion, under the influence of the above-mentioned factors, among which gravity plays a prominent although not the chief rôle, first accumulates in the dependent portion of the thorax, that is to say, in the complementary spaces of the lung between the crura of the diaphragm and the thoracic wall. This induces a relaxation of the diaphragm; the muscle descends and, owing to the presence of the fluid, part of the lung is withdrawn from the full influence of the diaphragm, just as the effect of the lung on the diaphragm is lost. Accordingly the inspiratory flattening and downward movement of the diaphragm will be diminished in proportion to the extent of the effusion separating it from the lung, and since, owing to the obliteration of the capillary space between the lung and the diaphragm, the traction exerted on the lung is interfered with, the expiratory contraction of the lung is diminished in the same proportion, because the overloaded diaphragm has to be elevated by the lung during expiration. In other words, the respiratory excursions of the lung and diaphragm diminish as the exudate increases, because the correspondence between the two factors working in the same direction is lost, or at least materially modified.

But the mechanical effect is not the only one exerted on respiration by the exudate, since it has already been stated that the degree of atelectasis bears no proportion to the extent of the exudate. Evidently some other influence is at work which, in my opinion, is more important than any other, although it has not received the attention it deserves in the discussions of this subject. I have been unable to find in the literature any reference to the important fact that the antagonism between the so-called elasticity of the lung which tends to contract the lung concentrically, and the intrabronchial pressure which tends to dilate it, is really an antagonism between the *tonus of the dilators* of the thorax and the *tonus of the contractors,* the most important element in which is the tissue tonus of the lungs, or, in other words, its power of retraction. The lung during life does not contract solely by virtue of its elasticity, any more than muscles extend by a relaxation of their elasticity or contract by an increase in that important function. The lung is not expanded by the traction of the muscles, *i. e.,* by distention; its volume is not increased by force, but solely by a *relaxation of the pulmonary tonus,* which occurs whenever enough air is forced in by the intrabronchial pressure to fill the vacuum produced.

The work of the dilators of the thorax, the inspiratory muscles, is almost entirely lost whenever for any reason the tonus of the lung at the beginning of inspiration refuses to relax, as in bronchial asthma, or when there is any obstruction to the entrance of air, as in stenosis of any portion of the air-passages. It is also a well-known fact that even a considerable diminution of the intrabronchial pressure (as when the atmosphere is rarefied) does not prevent respiration, even without undue exertion of the respiratory muscles, provided there is a corresponding diminution in the tonus of the lung at the beginning of inspiration, because the work required of the respiratory muscles for expansion is correspondingly diminished.

As will be shown more at length later on (see page 915) the lung, in common with all other tissues, possesses an active tonus, by virtue of which the organ, which is held within the thoracic cavity by the two layers of the pleura, becomes by a special arrangement of all its component parts tense in the physical sense without being distended. By this means the volume becomes enlarged by relaxation or *loss of tonus,* without distention, and diminished by an *increase of tonus,* again without distention.

This apparently paradoxical view becomes intelligible when it is remembered that the living tissue consists of two dynamically different portions: the *living* molecules which are capable of great cohesion, and the *dead* molecules or the molecules of tonus and water, so that any alteration in the volume is coupled with the expulsion or reception of fluid and a redistribution and dynamic union of the smallest elements.

The living tissue must, therefore, be made up of resting portions (not visibly in motion), capable of taking up (endowed with great powers of combination), and of working, actively expelling portions, in which visible motion is great and the power of combination small in proportion to the expelling power or that which performs visible work. In no other way is the arrangement of the parts on which tonus depends and the prompt performance of work with so little friction possible.

It is like a rope that is stretched taut by weights attached to each end. When subjected to an excessive strain, the rope would part, with the production of heat, if it were not moistened with water and a change in the distribution of the intermolecular forces thus brought about, so that instead of a solution of continuity, an additional shortening takes place.

There is a well-known, although not absolutely authentic story, that the erection of the obelisks in Rome by Fontana was accomplished by moistening the ropes with water, which not only prevented the heated rope from parting, but also furnished the necessary tension for the final lift.

The expansion of the lung during inspiration is not a distention like that, for instance, which takes place in a thin-walled hollow rubber ball. It is rather a relaxation due to loss of tonus, and this relaxation must eventuate in the filling of the lung with air whenever the dilatation of the thoracic walls allows distention to take place and the necessary air required to fill the vacuum is provided.

One of these conditions is fulfilled by the dilatation of the bony thorax through the inspiratory muscles which tend to contract under the influence of a maximum tonus and enlarge the intercostal spaces. The second condition is fulfilled by the simultaneous establishment of a free communication between the air-passages and the atmosphere, in which the openers of the glottis play the most important part.

Accordingly, there is an antagonism between the tonus of the inspiratory muscles of the thorax and the tonus of the pulmonary tissue. The inspiratory stimulus, conducted by the nerve-paths and the blood, is a double stimulus exciting the action of the inspiratory muscles and inhibiting the reflexly contractile portions of the lungs. And, conversely, the strongest inhibitory stimulus to respiration is an exciting stimulus to the pulmonary tonus, as is shown by the well-known experiments on the vagus and by distending the lung with air (Hering-Breuer and others).

Certain stimuli affecting the pleura tend to reinforce the expiratory stimulus, and since they work in the same direction as the pulmonary tonus they are at the same time inhibitory stimuli to inspiration. The most important of these stimuli is inflammation of the pleura, and accordingly the excursion of the affected half of the thorax is diminished by the accumulation of an exudate, and this diminution would be even greater were it not that the simultaneous reduction in the respiratory surface, which acts as a compensatory reinforcement of the inspiratory muscles, prevents an excessive lessening of the inspiratory movements.

Since, however, the stimuli have a mutual effect on one another, it is obviously very difficult to determine beforehand the exact state of the respiratory activity in the presence of exudations. There is no doubt that true dyspnea indicates a lessening of the respiratory surface, while a mere diminution in volume of one-half the thorax may be regarded as indicating irritation within the pleural cavity, acting as a stimulus on the tonus of the lung, and therefore having the effect of an inhibitory stimulus. But, in addition to this reinforcement of the effort on the part of the lung to contract, there is another factor at work in the production of local atelectasis due to the pressure of the exudate.

For, since the exchange of gases between the air in the lungs and the pulmonary vessels—the essential activity of the organ which we somewhat incorrectly designate absorption of oxygen, whereas it is in reality a specific, partly chemical and partly physical activity, in other words, a secretion—depends chiefly on the strength of the inspiratory and expiratory air-current or the power (kinetic value) of the moving current of air, a mere diminution or stoppage of the air-current in those portions which are chiefly concerned, namely, the lowest portions of the lung, necessarily favors the absorption of the remaining air by the pulmonary vessels, and possibly also by the surrounding pleural fluid. This, then, is an additional factor tending to favor diminution in the volume of the affected portion of the lung by the constantly increasing tonus,—the elasticity of the organ,—or, in other words, for the production of the condition which is known as atelectasis.

The same thing occurs in the other portions of the lung; for, as the fluid seeks a higher level with the increase of the effusion and forces itself between the upper portions of the thoracic wall and the lung, the same interference with the entrance of air necessarily takes place and leads to the production of absorption atelectasis, conditions which have been pointed out by Traube, and later more particularly by Lichtheim.[5]

It should be mentioned that the events just described may be brought about by any form of exudate, since the retraction of the lung depends on the strength of the intrapleural stimulus which reinforces the tonus of the lung and inhibits the tonus of the respiratory muscles. A considerable diminution in volume, or at least a restriction of the movements of one-half of the thorax, may, therefore, take place when the two layers of the pleura are separated only by a layer of fibrin, the product of intense inflammation, or become the seat of inflammatory hyperemia. In a word, the mechanism of the air-supply of the lung may be disturbed by anything which affects the capillary space between the two layers of the pleura, or, to be more precise, by any modification of the stimuli which are produced in that region and act on the extremities of the vagus.

A common result of large effusions is **dislocation of the liver,** either downward or in the form of an oblique dislocation of the transverse axis. In the former case the lower border is displaced in a line parallel to its normal position; in the latter the organ is rotated about the suspensory ligament as an axis, so that one lobe—usually the right, which is the heavier—is displaced downward, and the other—usually the left—upward.

These cases are not sufficiently explained by the simple statement that this peculiar lever-like action is merely the result of dislocation of the diaphragm. One is forced to assume that the conditions result from the action of certain dynamic influences, the effects of which may manifest themselves in different ways. The theory of a purely mechanical dislocation of the diaphragm is untenable, since such an event would necessitate complete suspension of the tonus and contracting power of the muscle, as well as of the tonus of the lung. The mechanism of *dislocation of the diaphragm* is generally as follows:

The needful space for the exudate is at first obtained by a certain dilatation of the thoracic space, achieved by relaxation of the (reflex) muscle tonus and distention of the other tissues of the thoracic wall. This is followed by a secondary compensatory diminution of that portion of the lung which is particularly affected, as has been described, while a true dislocation of the neighboring organs occurs only as the last link in the chain of events.

This dislocation by pressure is counteracted by a kind of counterpressure effected by a compensatory increase in the respiratory activity in other portions, in proportion to the reduction of the respiratory surface in the affected area, unless movement is inhibited by excessive pain. In this the compensatory activity of the other lung is the most important factor, and hence it is that in the great majority of cases, even when the exudate is so great as to displace the sound lung laterally, the corresponding thoracic muscles and sound half of the diaphragm act more vigorously, with the result of a downward displacement of the diaphragm on the sound side which prevents the rotatory dislocation of the liver about its axis by favoring dislocation of the organ *in toto*.

It finally remains to mention the **dislocation of the mediastinum.** Here, again, we have to deal, especially when the fluid is beginning to accumulate, not with a dislocation due to the direct pressure of the exudate,—in other words, a compression,—but with conditions exactly similar to those which have just been described. The exudate acts on the lung exactly like a tonic stimulus wherever it comes in contact with the walls of the cavity, and thus favors contraction. Hence all those portions of both lungs, which are subjected to these altered stimuli and not only to variations in pressure, contract actively, while other portions, owing to the inhibitory stimulus exerted on the tonus of the thoracic walls or the more powerful stimuli to inspiration brought about by diminution of the respiratory surface, undergo compensatory expansion, and the final resultant of these individual exciting and inhibitory impulses determines the position of the mediastinum and the central position of the diaphragm as well as the volume of the individual portions of the lungs which are either contracted or compensatorily expanded.

It may be incidentally mentioned that the difference in the breathing produced by the lagging behind of the diseased half of the thorax manifests itself particularly in an increased activity of the sound side.

We must not omit to advert to the interesting *manometric investi-*

gations of Schreiber, who found that the diminution in pressure at the beginning and end of deep inspiration may be much greater than has been stated by other authors (Leyden,[5] Homolle,[5] Quincke[5]). Thus, Leyden found with an initial pressure of +24 a fall during inspiration to +16 mm.; Homolle from +15 to +5 mm., while the difference determined by Schreiber was from +2 to —28, and even less. A similar variation is found in the figures for the final pressure: Leyden —28 to —42 during inspiration; Schreiber —40 to —90.

From these and other pneumatometric experiments Schreiber rightly draws the conclusion that pneumatometric and pleurometric figures, in patients suffering from pleurisy or affections associated with increased pressure in the thoracic cavity, represent the inspiratory or aspiratory activity not of the diseased, but of the sound half of the thorax, and that the manifestations of negative pressure on the part of the pleura of the diseased side are to be regarded chiefly as the expression of activity of the sound side. (Unfortunately the effect of alteration in the tonus of the diseased lung is left out of consideration in this explanation.)

Among experimental investigations on the consequences of diminution of space in pleurisy should be mentioned the work of O. Rosenbach, who studied the effect on dogs and rabbits of artificially increasing the pressure within the pleural cavity by the injection of oil, plaster-of-Paris, and air, or the introduction of a hollow rubber ball, and obtained somewhat different results from those of former investigators.

The important fact, first determined by Lichtheim,[5] that the **blood pressure** is not affected by considerable diminution in the volume of the pleural cavity, does not, as pointed out by Rosenbach, justify us in assuming that there is a similar constancy in the circulatory and nutritive processes corresponding to the constancy in blood pressure. The arterial pressure is maintained at the expense of an enormous amount of extra work on the part of the organs which have to make up for the mechanical obstruction to the circulation offered by the exudate, and although the main arterial pressure remains the same, there are local areas of venous hyperemia or arterial anemia.

The heart is, of course, chiefly concerned in supplying this extra demand, and shows its extra work by the great increase in the frequency of its contractions. In addition, there is a marked increase in the force and rate of respiration, and the caliber of the arteries is considerably diminished by the contraction of the muscular elements of their walls, all of which goes to show the great difference between the amount of work performed under normal conditions and that performed under the influence of increased pressure, and the difficulty—not to say impossibility—of complete local and general compensation.

The increased activity of all the organs, it is true, tends to compensate to a certain extent for the immediate deleterious effects of the diminution in the volume of the pleural cavity on the circulation by maintaining an average pressure sufficient to supply the chief demands of nutrition. But it is unable to maintain perfect compensation for any length of time because, although the pressure remains constant, there is necessarily a diminution in the rapidity of the blood-current and of the volume of the heart-beats which are necessary if all the tissues are to perform their allotted work.

Although the body is able for a short time to supply enough material for the work necessary to keep the organism together, it is incapable of performing any intrinsic muscular work, or even of supplying the nourishment required under ordinary conditions, as is shown by the immediate occurrence of dyspnea and diminution of the pulse following any excitement or movement on the part of the animal. The same thing is noticed at the bedside. Although the patient may apparently not be suffering from dyspnea and his pulse-rate may be normal, a marked disturbance of respiration and cardiac action will at once manifest itself whenever he makes the slightest movement, such as sitting up in bed, coughing, loud talking, etc.

Hence maintenance of the blood pressure does not mean a maintenance of the entire or even of the local work; and although the pressure may remain constant, there may be a considerable deficit in the blood-supply of all the organs, especially the brain. The anemia of the brain was clearly indicated in the experiments by the distinct irritation of the center of the pneumogastric and of the vasomotor center, showing itself in arrhyhtmia and retardation or intermittency of the pulse which occurred when the volume of the pleural cavity was diminished.

In attempting to explain the occurrence of sudden death in pleurisy with a large effusion when direct cardiac or organic disease can be excluded, we must consider the effect of obstruction to the venous flow, and particularly an obstruction of the vena cava by kinking (see page 880). The compression of this large venous trunk is not always due to a direct displacement of the vein by the exudate, but may also be produced merely by a strong contraction of the sound half of the diaphragm, which at every forced inspiration drags the vein over toward the sound side and almost obliterates the lumen, especially if, as happens when the exudate is large, the depressed, atonic half of the diaphragm (on the diseased side) is not functionating. This mechanism, as Rosenbach has shown, also plays the chief part in the production of the *pulsus paradoxus*, or inspiratory diminution of the pulse, as any increase in pressure within the thorax by the presence of a fluid leads to a temporary interruption of the venous flow during inspiration, and this interferes with the proper filling of the chambers of the heart.

It is quite possible that the dysphagia and the occasional paroxysm of singultus are due to the effect of this spasmodic action of the diaphragm on the mediastinal structures, especially the esophagus.

VIII. GENERAL SYMPTOMATOLOGY.

Modes of Onset.—The development of pleurisy manifests itself in so many different ways that it is impossible to draw a typical picture. In a large number of cases, even in vigorous individuals with good powers of reaction, it may develop so slowly and painlessly that it is some time before the patient is led to seek medical aid for the general malaise, slight dyspnea, loss of appetite, and general loss of strength.

It is, therefore, important whenever the patient complains of digestive troubles combined with marked loss of appetite, general depression, chilliness, and the rapid development of pallor, to subject the lower and lateral portions of the lung to a careful examination. A slight dulness can often be elicited in this region at a very early stage, and this suffices to establish the diagnosis.

An early diagnosis is particularly desirable in pleurisy because the simplest hygienic treatment, absolute cessation of work and rest in bed with appropriate diet, yields the most satisfactory results. There is no doubt whatever in the author's mind that in acute pleurisy, as in typhoid fever and acute articular rheumatism, those cases which are recognized early and immediately withdrawn from all harmful influences run a distinctly more favorable course than the neglected ones.

In other cases pleurisy, like pneumonia, is ushered in by a *severe chill.* There are severe pains in the chest and back, cyanosis, and not rarely distressing cough without expectoration. If the cough is severe, the sputum is often streaked with blood, but it does not show the characteristic color of pneumonia sputum.

A third class of cases present chilly feelings, especially in the back, along with more or less severe general pains. There is a slight evening elevation of temperature with profuse sweating.

Fever.—Pleurisy does not exhibit a typical fever curve. I have seen a number of cases where the most careful registration not only failed to show a rise in the temperature [especially in old subjects.—Ed.], but in which the temperature was even subnormal, and others, again, where the temperature for several weeks attained its maximum, which was not always high, at various times of the day. On the other hand, cases are not rare in which there is a continued fever as high as 41° C. (105.8° F.), with intercurrent chills, during the first weeks. *Hectic fever* is not rarely observed even in non-tubercular cases, both during the accumulation of the exudate and during the stage of absorption when the latter is very slow. A temperature above 40.5° C. (104.9° F.) in the axilla means much more in pleurisy than in pneumonia, especially if the curve is of an inverse type, with the highest register at noon or during the early hours of the day. With a *purulent exudate* the fever is usually uniform, continuous, and high; with *putrid exudates* it is more apt to be irregular and interrupted by chills and a period of subnormal temperature following the chills.

In any case the occurrence of *profuse sweats* exerts a marked influence on the fever curve. Accordingly, the fever is often remarkably low in putrid effusions, the temperature, as has been mentioned, being at times subnormal, especially when the distressing symptom of profuse sweating is well marked.

A *terminal pleurisy*, and the peculiar form of right-sided pleurisy which is most frequent as a concomitant of diseases of the liver and cardiac weakness, usually have no effect on the temperature curve. In general, it may be said that in debilitated individuals pleurisy may run an absolutely afebrile course.

In such cases it goes without saying that even a normal temperature is as significant as a rise in temperature in other cases, especially if the drop is not accompanied by improvement of the general condition and of the appetite. In cachectic and anemic individuals a morning temperature of 37° C. (98.6° F.) with an evening temperature of 37° to 37.5° C. (99.5° F.) is to be regarded as febrile and as a certain sign of beginning terminal pleurisy or pneumonia.

In general, an increase of the exudate is associated with a rising temperature or a continued form of fever, and a diminution in the exudate with irregular temperatures or a normal curve. But the temperature curve depends so much on the basal disease, as well as on other conditions that prevail in the individual case, that this statement cannot be accepted as a general truth.

A rise in temperature without any physical signs indicating an increase in the exudate is particularly apt to occur in relapses of tubercular pleurisy or bronchopneumonia, in marked hyperemia of the pleura, or when there is an abundant secretion of fibrin with the formation of membranes. The rise in temperature is often accompanied by a diminution of the exudate, and this contrast between the physical signs and the temperature is a bad prognostic sign, especially when the appetite is diminished.

The occurrence of a so-called *resorption fever* recorded by a number of authors is not without some clinical foundation. No definite proofs have, however, been brought forward. There is no doubt that an unusual rise in the temperature representing a kind of crisis is often followed by sudden diminution of the exudate, and a case can never be regarded as cured, even when the patient's general health appears to be restored, until the temperature has permanently fallen below the normal.

The afternoon and late evening temperature is of special importance, and I may remark incidentally that temperature, appetite, and sweating are often more important points in the prognosis than the signs elicited by physical examination, which are necessarily slow to show any change once the exudate has materially diminished.

Defervescence usually begins in the second or third week; it is at first very inconsiderable, and manifests itself only in the morning hours; later a fall is observed during the evening hours, and toward the end the fever curve becomes quite irregular with slight afternoon and evening rises.

The difference in temperature between the sound and the diseased side, insisted on by various authors who state that the temperature is always higher in the axilla of the diseased side, is not by any means constant, although it cannot be denied that in many cases of pronounced pleurisy a higher cutaneous temperature prevails over the diseased half of the chest.

Cough.—It is remarkable how often cough is absent in pleurisy; in most cases the cough, which is often distressing, is observed only when the patient sits up and draws a deep breath, being caused by the expansion of collapsed portions of the lungs or the irritation of the bronchial mucous membrane in the deeper portions of the lung by sudden contact with the air. On the other hand, slight bronchial irritation and *hawking* are much more frequent.

Violent paroxysms of cough are also observed when large masses of exudate are evacuated by operative means; these paroxysms may with propriety be explained by Hering-Breuer's experiments, in which any sudden expansion of the lung, or, to speak more precisely, every maximal inspiration, is followed by a correspondingly strong expiration as a result of sudden expansion of retracted portions of the lung.

It may be that the increased amount of arterial blood supplied to certain portions of the lung during the evacuation of an exudate acts as an expiratory stimulus by stimulating the vagus. The blood-vessels after a long period of occlusion are often stimulated to excessive activity by the sudden inflow of blood, so that an exudation occurs into the alveoli and symptoms of pulmonary edema make their appearance. Hence the severe expiratory movements may have a certain advantage, inasmuch as they retard the flow of blood to the lung and remove any tendency to hyperemia by assisting in emptying the blood-vessels.

The cough may, of course, be due to concomitant processes in the lung instead of directly to the pleural affection, and its severity is accordingly in proportion to the irritation of the pulmonary parenchyma and sensitive portions of the bronchial mucous membrane.

According to Nothnagel's[5] experiments, which do not quite agree in their results with those instituted by Kohts,[5] irritation of the pleura in animals is not followed by cough. It is also well known that after an empyema operation irritation of the pleura (which, it is true, is diseased and has therefore suffered a diminution of its power of reaction) by contact with the hand or a surgical instrument is not followed by cough, any more than the mechanical irritation of the trocar-cannula which is brought in direct contact with, and rubbed to and fro on, the pulmonary or diaphragmatic pleura, as any one can convince himself during an exploratory puncture.

Sputum.—The production of sputum in pleurisy is obviously a secondary process. The bronchi of the retracted lung can be irritated only when there is an independent disease of the lung or an occlusion of the air-passages with the production of a sparse, tenacious, mucous, and rarely purulent sputum.

If a pleural effusion ruptures, or filters through into the lung, as it is termed, either by direct perforation into an open bronchus or through a necrotic area in the pulmonary pleura into the loose alveolar tissue, an event which is rarely followed by complete anemia of the thorax, large masses of sputum are expectorated, consisting usually of purulent or tenacious mucopurulent masses of greenish-yellow color and musty or even fetid odor.

Occasionally an abundant expectoration is observed in the absence of perforation, when there is marked compression of the lung and the pressure is temporarily raised by occlusion in the distribution of one of the larger bronchi. In such cases a temporary bronchiectasia is formed which may be quite considerable and is due to relaxation of the tonus of the bronchial muscles. Within this dilatation of the bronchial lumen large masses of greenish-yellow pus accumulate which are evacuated from time to time, when the internal pressure has reached a certain degree, probably by a maximal tonic contraction of all the bronchial and expiratory muscles such as usually occurs only after a distressing paroxysm of cough.

I have seen many cases in which all the symptoms during life indicated that no communication could have existed between the exudate and the bronchial system, and the postmortem confirmed this supposition, so that one is forced to conclude that the so-called "*mundvolle*" (copious) expectoration does not necessarily indicate rupture of an exudate unless the physical and functional conditions in the pleural space after expectoration undergo a material change; that is, the respiratory murmur clears up, the dulness diminishes, or pneumothorax develops. The latter event is a surer sign that rupture has taken place than are the two others, for since after a copious expectoration even those parts of the lungs which were collapsed as the result of severe cough temporarily expand and functionate, mere diminution in the dulness and return of the respiratory murmur cannot always be directly attributed to the evacuation of the pleural fluid.

The dulness and diminution of the respiratory murmur produced by temporary *bronchiectatic processes* are never as marked as the dulness produced by an exudate or tissue changes in the lung; hence the difference in the physical signs before and after the evacuation of bronchiectatic sputa is relatively slight. Fetid sputum and sputa containing numerous fatty acid crystals and elastic fibers occur practically only when a putrid exudate is discharged into the bronchi, or in chronic putrid bronchitis, which, in contradistinction to gangrene of the lung, is almost never associated with pleurisy.

Pain.—The pain of pleurisy occasionally attains extraordinary severity. It may be cramp-like and colicky, radiating to both sides of the vertebral column and into the arms, shoulders, and epigastrium, especially in diaphragmatic pleurisy. In *dry pleurisy* the sensations experienced usually resemble muscular pains; they are dull, stabbing or tearing, and reach their maximum at the end of inspiration; *with a large exudate* they are usually dull and diffuse, and become very much aggravated by movement, especially coughing and sneezing. External pressure aggravates the pain, particularly when the muscles and the pleura are much involved, and the intercostal depressions are obliterated so as to permit of deep pressure. *Pain on pressure,* however, is never as severe as in phlegmonous inflammation of the muscles, periostitis, pericarditis, or peripleuritis.

The pain of pleurisy not infrequently simulates *intercostal neuralgia* with distinct points of sensitiveness along the vertebral column and the course of the nerves, associated with considerable hyperesthesia of the skin. In very rare cases there may even be pain on the sound side, usually confined to a circumscribed area. [I am in attendance with Dr. Morton upon a patient aged eighty-four, who presents the coincidence of pleurisy and intercostal neuralgia or neuritis (ninth nerve), as the characteristic eruption of herpes appeared along the course of the intercostal nerve and on the side of the pleuritis. In the first week of illness the pleurisy with friction sounds, pain, and fever dominated; during the second, third, and fourth weeks the intercostal neuralgia was most marked. The latter was not influenced by respiratory movements, was distributed to the epigastrium, and in the third week the eruption appeared.—Ed.] This form of radiation has never been sufficiently explained; the fact that the terminal fibers of the intercostal nerves decussate and intertwine in the median line does not, in our opinion, serve to explain the occurrence of pain on the other side in the areas of distribution of all the nerves.

The pain of disease of the vertebral column in tumors, caries, etc., is usually distinguished from pleuritic pain by its greater intensity, its constancy, and the absence of distinct localization. Another characteristic feature is that it is aggravated by the slightest movement.

The pain, except in rare cases, is probably not due to the direct pressure of the exudate on the nerve-trunks, but rather to unusually severe and wide-spread irritation of the nerve-endings, or to the distention of the vessels and excessive engorgement of the lymph-spaces with cells and fluids; in other words, to active and passive hyperemia.

Headache is not an uncommon symptom in pleurisy; it is usually due to the fever and, when the exudate is great, to the interference with respiration or to venous stasis.

The symptoms referable to the **digestive apparatus** may, as has already been mentioned, be quite severe even in cases which run their course without any other symptoms. When the patient is decidedly ill, anorexia and increased desire for water follow as a matter of course, especially if the fever is high; but it may be said in general that the slower the course, the more intense the digestive disturbance and the consequent anemia. Retching and vomiting are often produced by mechanical irritation of the structures concerned. The mere encroachment of an extensive exudate on other organs may suffice to produce digestive disturbances on account of the interference with the mechanical ingestion of food, and the consequent compression and dyspnea following a hearty meal.

Ferber[6] has observed that in very rare cases of diaphragmatic pleurisy the descending bolus of food produces marked pain at the moment of its passage through the esophageal foramen. I myself have seen deglutition interfered with in many ways in various cases, but it should be remembered that pain in the sternal and deep pharyngeal muscles alone, such as occurs in muscular rheumatism and from other causes, may produce dysphagia of precisely the same character and equal severity.

Singultus is a frequent concomitant of severe forms of pleurisy, and I have often observed it even in very mild cases. It is probably due in most cases to direct irritation of the diaphragm, more rarely to irritation of the trunk of the pneumogastric or phrenic, and accordingly constitutes one of the most important symptoms of diaphragmatic pleurisy. In debilitated individuals it is a very ominous symptom.

Constipation.—Sluggishness of the bowels is readily explained partly by the diminution of the amount of food ingested, partly by mechanical causes, such as altered position, and the cessation of muscular movements, especially of the abdominal muscles and the diaphragm, which is most marked in painful cases.

[**The Blood.**—Morse* reports 224 separate blood counts, taken from primary cases exclusively, in only 13 of which the number of leucocytes was above 10,000. Of these counts, 9 belong to the same case, one due to pneumococcus infection.

The author concludes that the leucocytosis bears no relation to the temperature; to the character of the fluid, whether bloody or containing few pus-corpuscles; to the amount of the fluid; or to variations in the amount of the fluid, as, *e. g.*, after and before tapping, and during reaccumulation.

The condition is valuable in two ways. (1) If the physical signs are doubtful, and there is no leucocytosis, the case is probably one of serous pleurisy, and not of pneumonia or empyema. (2) If serous pleurisy is present and there is marked leucocytosis, it is probable that a complication exists.—Ed.]

Urine.—The urinary secretion in pleurisy is the same as in other febrile diseases except that a very large exudate and weak cardiac action exert a direct mechanical influence. Under such conditions the activity of the kidneys is reduced to a minimum, partly by the positive pressure of the exudate in the thoracic cavity interfering with the venous flow, partly by the diminution of the respiratory movements and the consequent paucity of oxygen, and finally by the diminished arterial tension. As the amount of urine secreted diminishes, the percentage of albumin and salts increases, so that all grades of albuminuria are observed.

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* *Am. Jour. of the Med. Sci.*, November, 1900.

The *albuminuria* is due to venous stasis, to the fever, and possibly to the absorption of certain substances which irritate the kidneys. Thus the albuminuria observed in empyema with high pressure is probably due to direct absorption from the purulent focus. In the same way the peptone that is occasionally found in the urine may be regarded as a product of absorption (in empyema), and some have even gone so far as to accord this substance a certain diagnostic value for empyema, a method of diagnosis which, to say the least, is much more inconvenient and unreliable than exploratory puncture. The albuminuria may, of course, also be regarded as a so-called regulating albuminuria (Rosenbach) necessary for the disposal of albumin which, owing to the morbid process, has failed to enter into combination in the blood, and can, accordingly, not be utilized in the organism.

Morphologic constituents are almost always absent in the urine in uncomplicated cases. An abundance of pus-corpuscles and blood-cells or cylindric epithelium points directly to the presence of a purulent effusion or to inflammation in other organs, or to sepsis.

During the period of absorption a marked increase in the urinary secretion is generally observed. This increase rarely precedes a decrease in the amount of urine; the order of events is more frequently reversed. It is a sign that the abnormal urinary secretion due to the exudate is about to subside, but the factors to be considered in this connection are so various that the true cause for this rise in the urinary secretion is difficult to explain in individual cases. It appears certain, however, that the mere relief from the mechanical pressure of the exudate plays a relatively minor part.

Sweating.—With the exception of articular rheumatism, there is no other disease in which excessive sweating is as common as in pleurisy. The more intractable the fever and the longer the duration of the febrile period, the more common is the occurrence of sweats during the diurnal or permanent period of defervescence. In moderately severe cases running on for weeks without any improvement in the symptoms there may be veritable paroxysms of sweat recurring several times a day, to the great detriment of the patient's strength. These paroxysms must be regarded as a mild form of hectic sweats, such as occur frequently in pyemic processes, in latent suppuration, and in phthisis.

During the early stages of pleurisy the sweating merely represents the normal reaction of the organism to the excessive febrile irritation; later on in the disease, however, it probably indicates a vasomotor paralysis of the nerves supplying the vessels in the skin, hence these excessive sweats are frequently a sign of weakness in purulent and tuberculous pleurisy.

There is, of course, a reciprocal relation between the urinary secretion and that of the sweat-glands, and if the patient is sweating copiously, a diminution in the amount of urine does not necessarily indicate that either the heart or the kidneys are beginning to weaken, especially if albumin and morphologic elements, and particularly red blood-cells, are absent from the urine.

Pulse.—The pulse-rate and the condition of the arteries in uncomplicated cases almost always correspond to the fever curve, unless there has been some unusual excitement; later on the quantity and quality of the exudate exert a certain influence on these factors. The greater the exudate, the higher, as a rule, the arterial tension and the pulse-rate. Retardation of the pulse-rate is often a sign of irritation of the heart-muscle or interference with the venous flow from the cranium and, like arrhythmia, should receive careful attention, especially if combined with a narrowing of the caliber of the arteries. A slow pulse in the beginning of the disease is often a good sign and indicates vigorous heart action; the arterial system is usually at the same time well filled (hyperdiastole of the heart). The pulse, for obvious reasons, is considerably influenced by the pain, the general condition of the patient, and the particular kind of respiratory disturbance.

The various factors influencing the pulse-rate and **arterial tension** have never been fully explained, because the reflex alterations of protoplasmic activity brought about by the action of the poisons localized in the pleura have been too much neglected and because of the difficulty of measuring the reflex irritation of the vasomotor center on which the local arterial tension largely depends.

That the diminished volume of the pulse is not always due to mere obstruction of the pulmonary vessels by the exudate, and consequent diminution of the amount of blood going to the left heart, is not to be denied, although we cannot quite accept Lichtheim's experiments as conclusive in this respect, because they do not sufficiently reproduce the clinical conditions and only take account of the alterations in the pressure in the large arteries, such as the carotid or femoral, to the exclusion of all the smaller vessels.

Lichtheim [5] attempted to show that, owing to the peculiar character of the pulmonary vessels which possess little or no tonus, ligation or obstruction of almost three-fourths of the entire vascular channel is not followed by any diminution in arterial pressure, from which he reasons that this phenomenon, which, it is true, is *à priori* unexpected, can only be due to the fact that the same amount of blood flows through the remaining portion of the lumen as originally passed through the entire cross-section. He accordingly attributes the obstructing action of an extensive exudate not to occlusion of the pulmonary arteries, but rather to a displacement and compression (kinking) of the large vessels and to direct pressure on the heart.

While I am inclined to agree with the latter conclusion, I cannot subscribe to the former one; for, even admitting that an equal degree of blood pressure corresponds to an equal amount of blood-supply and to the productive work of lungs and protoplasm, the experiments are based solely on temporary phenomena. But temporary compensation is a very different thing from permanent accommodation, and though compensation may apparently be established in a resting animal, this does not prove that the organism is capable of performing even a fraction of the work necessary for the various demands of life under such altered conditions.

Hence although a perfectly sound heart might be able to perform its work for half an hour after occlusion of all the pulmonary vessels, this excessive amount of work would of course be impossible in a disease which may extend over days, weeks, or even months, and the mechanical obstruction of only a part of the lungs may easily bring about a marked disturbance in the circulation, even though the mean pressure in one of the larger arteries may not at once show any diminution, or may even become increased for a time owing to constriction of all the peripheral vessels which results from a diminished blood-supply to the periphery.

Respiration.—It is evident that the oxygen-supply of the body, and hence the **respiratory rate** and the **color** of the patient, are directly dependent on the specific mechanical and chemical work of the lung and circulatory apparatus; but it is equally evident that these two conditions are also largely influenced by the original strength of the individual, the amount of blood, the rapidity with which the exudate accumulates, and particularly the amount of pain experienced. The respiratory rate especially depends more on the variety of pain present than on the effect of fever and the extent of the exudate. Any dry pleurisy, even without much rheumatic pain in the thoracic muscles, may be accompanied by a high grade of dyspnea if the pain in the thoracic muscles is sufficiently great.

The respiration in such cases is quite characteristic, being extraordinarily short, spasmodic, labored, and sighing—characters that are directly due to the pain. The patients are often extremely depressed and markedly cyanotic. These phenomena are particularly marked in plethoric individuals and often assume alarming proportions; the amount of cyanosis and turgescence of the countenance seems to indicate a much more grave interference with respiration than really exists. As the strength gradually diminishes owing to the rapid increase in the exudate and the failure of absorption to take place, cyanosis as a rule becomes less conspicuous than dyspnea. The respiratory rate is comparatively low, the breathing becomes almost slow, expiration particularly being surprisingly deep, although it of course requires the co-operation of all the accessory respiratory muscles.

The greater the depression of the diaphragm, or, to speak more correctly, the greater the inhibition of its activity owing to the loss of tonus, the more marked is the dilatation of the lower portions of the thorax and the limitation of respiratory movement in this region. There is usually a marked contrast between this phenomenon and an excessive dilatation of the upper part of the thorax on one or both sides.

There is hardly any loss of *strength* in dry pleurisy, even in cases where the disease begins with high fever and, after a stormy initial period, complete absorption ending in relatively rapid recovery, takes place. The insidious forms of inflammation, on the other hand, especially tuberculous and plastic pleurisy associated with hectic fever and colliquative sweats, are often accompanied by rapid loss of strength. The greater the disturbance of the digestive apparatus, the more rapid the development of high grades of *anemia*. Other factors which lead to rapid loss of strength are excessive pain, insomnia, hemorrhagic exudate, and the occurrence of chills.

In many cases the patient's **attitude** is characteristic of the variety of disease present. The greater the pain in the diseased side, the more irresistible the tendency on the patient's part to assume the dorsal position or a kind of diagonal position with the convexity of the body toward the diseased half. If the exudate is very large and attended with much pain, the patient almost never lies on the sound side, because the breathing would be too much interfered with in this position. In such cases the patient preferably lies either on his back or on the diseased side. As the interference with respiration or the encroachment on the respiratory surface increases, the patients tend more and more to assume the dorsal or even a sitting position, on account of the greater ease, in this attitude, of expanding the entire thorax, particularly the upper portions.

IX. SPECIAL SYMPTOMATOLOGY.

1. INSPECTION.

On inspection a certain characteristic **retraction** of the inferior lateral portions of the thorax is observed. This is particularly well marked when the intercostal spaces are wide, and indicates a lack of expansion in those regions when the respiratory movements in the upper portion are increased, so that the latter is completely filled during inspiration. Retraction may, however, also be due to a circumscribed *adhesion* between the two layers of the pleura, or to *atelectatic conditions* and *consolidations*, because the pressure of the external air during inspiration effects a depression of the soft outer parts over these areas.

If there is much **pain,** this will be indicated by a characteristic inclination of the body toward the diseased side and a peculiar, accelerated, jerky respiration.

As the exudate increases the **shape** of the corresponding half of the thorax undergoes marked changes, the circumference of the thorax as measured by means of the cyrtometer or tape-measure shows an increase, causing the lower lateral portions to bulge, while the intercostal depressions become obliterated and the spaces between the ribs increase in width or may even bulge outward, as is readily determined by comparing them with the intercostal spaces of the sound half. It is important to measure the circumference during expiration, or while the patient is holding his breath, as the conditions are not as clearly brought out during inspiration; for if the pulmonary function is good, the upper portions of the thorax, even in the beginning of the disease, are well dilated by the compensatory activity of the respiratory muscles, and the characteristic barrel-shaped deformity is not marked except during inspiration. Later this difference tends to disappear more and more.

In many cases the **diaphragm,** when at its deepest position,—namely, during inspiration,—may be felt below the costal margin. Some authors affirm that it may be palpable in rare cases as a permanent downward bulging; personally I have never seen such a condition. [Absence of Litten's phenomenon or the shadow of the diaphragm is another valuable diagnostic sign. It may be particularly useful on the right side, to distinguish between pleural effusion and enlargement of the liver or subdiaphragmatic abscess, as the phenomenon is preserved in the two latter conditions.—Ed.]

The shoulder of the diseased side in cases of moderate effusion is often higher than that of the sound side, and the vertebral column, which at first shows a scoliosis toward the sound side, gradually tends to straighten out as the healthy lung becomes more vigorous in action; it may even show a convexity toward the diseased side. [In children, and in cases of large effusion in adults, a striking feature is the increased distance of the angle of the scapula from the spinal column, on the affected side.—Ed.]

The results obtained by inspection of the **heart** vary according to the position of the exudate, the degree of cardiac activity, the general state of the individual, the mobility of the lung, and the presence of adhesions. True dislocation of the heart is usually observed in cases of extensive, free exudates or in moderately large, encapsulated effusions in the left half of the thorax.

In the beginning the apex-beat disappears or a peculiar undulation is observed in the intercostal spaces of the precordial region, which, so to speak, collapse with systole. The impulse over the sternum and the area lying to the right of the sternum cannot always be referred to dislocation of the heart; as a rule, it merely indicates excessive action of the right ventricle, or either an active or a passive dilatation of the cavity. Passive dilatation may be assumed when the venous pulsation is marked or other phenomena of venous stasis are visible.

A strong venous pulsation does not necessarily indicate the presence of an extensive exudate, as it may be due to labored respiratory movements, on account of which the effect of the cardiac pulsation on the venous system is more perceptible than under normal conditions. Undulation of the veins with distinct engorgement during inspiration is a much more reliable sign of the so-called diminution of space in the pleural cavity than are variations in pulsation.

The **outer walls of the thorax** rarely present any visible alterations except in very extensive serous exudates leading to considerable interference with the venous flow from the thorax walls. Under such conditions there may be an increased resistance to the touch over circumscribed areas in the diseased side, which is particularly noticeable when folds of the skin are picked up between the fingers. As a rule, the only visible difference is that the cutaneous veins are more conspicuous and more tortuous than normal.

Edema of the skin is often observed in purulent effusions and in serous effusions that have developed very rapidly. The value of this phenomenon as a diagnostic sign of suppuration was formerly much exaggerated. [To support this view, with which we are in accord, we have seen a local edema over the right base in cancer of the lower lobe of the right lung.—Ed.] Edema due to venous obstruction may be observed in any debilitated individual, and the edema is not necessarily on the side of the exudate; it is usually observed in any part of the body, which, owing to the forced position of the patient, is especially exposed to pressure. Thus the edema is often very marked on the sound side, especially over the back and buttocks, and almost always indicates a distinct loss of strength.

A conspicuous phenomenon that may be observed even in moderate degrees of effusion, particularly when combined with great sensitiveness of the thoracic walls, and is almost certain to be present when the exudate is extensive, is a certain *asynchronism of the respiratory movements*, both the inspiratory dilatation and the expiratory contraction being perceptibly delayed on the affected side. Of course, similar appearances may be produced by dense adhesions of the lung or the early formation of atelectasis, as by a very large exudate, but in the latter case the absolute dilatation of the thoracic cavity is usually greater.

This asynchronism cannot be due solely to the interposition of an exudate between the lung and the thoracic wall, causing a delayed response to the pull of the thoracic muscles, since the phenomenon is also observed during inspiration and when the exudate is so small as to be almost imperceptible. We evidently have to deal with a reflex inhibition of respiration,—a motor impulse,—the crest of the wave, so to speak, being reached more rapidly on the sound than on the diseased side. There is no doubt at least that the muscular contractions on the sound side are much less effective in advanced cases.

During the **period of resorption** in typical cases the symptoms reappear to a certain extent in inverse order, the tension of the thoracic wall and the edema diminish, the contractions of the respiratory muscles and of the diaphragm begin to reappear over the lateral portions, the apex-beat becomes palpable, and the diffuse cardiac impulse regains its full extent to the left of the sternum.

It goes without saying that a diminution in the circumference of one-half the thorax does not necessarily indicate absorption of the exudate, as it may be due to shrinking or dislocation of the organs. To determine whether it is really due to a diminution of the exudate the respiratory excursions should be carefully observed, for if the fluid exudate is undergoing absorption they should become more distinct as the lung regains its power of expansion; nor is the return of the cardiac impulse and apex-beat a certain sign that resorption is taking place. It may only indicate a dislocation of the heart by contracting adhesions, and should therefore be utilized with a certain degree of caution. It is important to determine, if possible, whether the apex-beat is accompanied, as under normal conditions, by a bulging of the intercostal space, or by a systolic retraction, as in the latter case it is more likely that adhesions have formed or that dislocation of the organ has taken place.

In very rare cases dislocation of the heart persists after normal conditions have been restored because the organ is held fast by dense adhesions, but even under such circumstances cases have been observed in which, after a time, owing to sudden or gradual division of the membranes, the organ returned to its normal position.

Certain deformities of the thorax, observed when there is defective adaptability of the lungs, are very characteristic. They may be due to deficiency in the degenerated and contracted muscles of the thorax, loss of elasticity of the ribs, ossification of the articular surfaces, atelectasis of the lungs, and dense bands of adhesion.

Abnormal curvatures of the thoracic vertebral column, with the convexity directed usually to the diseased side, are always compensated for by curvatures in some other portions of the column. A scoliosis in the upper thoracic region is regularly accompanied by one or more curvatures in the opposite direction in the lower thoracic and lumbar regions. The intercostal spaces may be contracted to such an extent that the ribs overlie one another like tiles on a roof. The scapula of the diseased side becomes displaced and projects from the thorax like a wing; at the same time it is usually, though not always, lower than that of the sound side.

The respiratory movements and respiratory murmur may be completely absent over the diseased lung, while the activity of the healthy lung is very much increased, and may show all the signs of emphysema.

2. PALPATION.

Palpation completes and confirms the results obtained by inspection. It affords a means of determining the strength of the costal muscles and the elasticity of the ribs, as well as the exact extent of the respiratory movement in different regions. By means of palpation we can also determine whether the periodic pulsations or bulgings of the chest-wall, corresponding to respiratory expansion and contraction, emanate from the heart and vessels or from the pleura itself, as in the latter case (*empyema pulsans*) they are usually divided by a non-pulsating zone from the area of greatest cardiac pulsation.

Palpation also reveals the presence of a **pleuritic rub,** and the ease with which this sign may be recognized will depend on the roughness of the pleura on which the vibrations depend and the power of the thorax to transmit the vibrations. If there is much subcutaneous fat and the chest-walls are soft and yielding, the friction fremitus cannot usually be determined by palpation, even though marked friction sounds may be heard on auscultation.

By palpation we also determine the **degree and seat of pain,** the position and resistance of the apex-beat, the dislocation of the heart, and the degree of displacement of the lung. In retraction of the left lung it also enables us to determine an important sign,—the so-called *diastolic click of the pulmonary valves,*—a phenomenon which possibly may emanate from the pulmonary artery, but certainly not from the valves, and is due simply to an alteration in the blood-current within the pulmonary arteries or to something which facilitates the transmission of the vibrations from that point. Both factors are favored by retraction of the left upper lobe, and also to some extent by the presence of infiltration in this region.

Fluctuation may rarely be determined when the exudate is very extensive, the intercostal spaces are very wide, the muscles flaccid, or when there is a complete absence of subcutaneous fatty tissue and of edema. The sign is present only in the lower lateral portions of the thorax, and is best elicited by pressing one hand firmly against the lateral wall, while with the other the lowest portion of the dorsal surface is percussed with moderate force.

Palpation also enables us to test the **pectoral fremitus,** which is a sign of some value, providing the many possible errors are carefully avoided. Among the sources of error the most important are edema of the thoracic walls, pleural adhesions, atelectatic and hypostatic processes, and, above all, occlusion of the bronchi by swelling of the mucous membrane or the presence of mucus, conditions which modify the sign, in the same way as a pleuritic effusion, by interfering with the intrabronchial transmission of the sound-waves.

Since atelectasis and the accumulation of mucus in the larger bronchi are the most important factors in the diminution of the pectoral fremitus, it should be observed as an invariable rule never to test the pectoral fremitus without asking the patient to draw several deep breaths and to clear the lungs by repeated coughing.

If the test is made immediately after the patient has assumed a sitting posture, the results are always very indefinite and unreliable. The tactile fremitus, which at first appears to be entirely absent over a large area, becomes clearly perceptible in different portions of the thorax at the end of a long examination. Relatively rapid and considerable changes in the fremitus may be occasioned by a rapidly sinking exudate or by layers of fibrin, hence in palpation, as well as in auscultation and percussion, the precaution should be observed of allowing the patient to sit up for several minutes before making a final examination of the dorsal surface, or at least to repeat the fremitus test at the end of an examination and compare the two results. The fact that the fremitus is normally somewhat more marked on the right than on the left side must not be lost sight of.

In apparent contradiction to this condition, which, according to our view, is the rule, Traube [6] frequently found that during a long examination the fremitus periodically vanishes and reappears, and he assumes that, owing to the constant tendency to atelectasis, the more accentuated expirations associated with phonation increase the collapse of the lung and thus diminish the fremitus until it is restored by the repeated deep inspirations.

But this contradiction is only apparent, since, owing to the complex and inconstant nature of the determining factors, the results necessarily vary from moment to moment, and do not follow any rule that can be determined beforehand. In many cases a rapid change in the phenomena is more conclusive than the absolute result of the first examination.

As a rule, in moderately large exudates *three distinct zones* can be distinguished on the dorsal surface, presenting marked variations in the fremitus. Over the lowest portion it is diminished or absent, becoming more and more distinct in the higher portions or even increased in a certain area, while, finally, the third or highest zone always presents a normal fremitus. This division into three distinct zones is readily explained on physical grounds.

The lowest zone corresponds to the region occupied by the thickest layer of fluid which has led to displacement and retraction or repression of the lung. The central zone of increased fremitus corresponds to that portion where very little fluid intervenes between the lung and the thoracic wall; but, as the retracted lung has become more or less atelectatic, it transmits the vibrations of the voice more readily, providing the bronchi are free. The highest zone, where the fremitus is normal, corresponds to the area where there has been no alteration in tension and in the amount of air contained in the lung.

Even when the entire half of the thorax is filled with fluid there may be a moderately large area in the interscapular space, close to the vertebral column, where fremitus is distinctly preserved. In this area the lung, although completely retracted, is not compressed and lies in close apposition to the thoracic wall, or else there is a large bronchus that is relatively superficial.

The fremitus is not always the same at every point on the same transverse line or at different times. Its strength depends on a variety of important local factors, and the fact that it varies in two adjacent areas does not by any means justify the assumption of encapsulation or of the presence of foci in the lung. Hence it does not seem to us permissible to base the diagnosis of *multilocular* *pleurisy* on difference in the fremitus, and for this condition also exploratory puncture is a much more trustworthy diagnostic method.

3. PERCUSSION.

Percussion is one of the most important means of diagnosis in the hands of the practised examiner, provided he does not interpret his findings according to set rules. It is, of course, valuable only when there is a fairly large exudate, and one should carefully guard against falling into the error of supposing that any marked dulness in the lower portion of the thorax, in the absence of râles and diminution of the respiratory murmur, indicates the presence of an exudate. In certain conditions of the thoracic muscles attended with pain, as *muscular rheumatism* or *intercostal neuralgia,* and even in certain mild cases of dry pleurisy, spasmodic contraction of the thoracic muscles often simulates dulness which promptly disappears after the pain has been allayed by an injection of morphin.

In doubtful cases, where a questionable dulness of this kind over the posterior and inferior portions of the pleura is present, a careful examination of the lateral wall of the thorax offers one of the most reliable means of determining the presence of a fluid exudate; if the exudate is on the left side, the area between the fifth and eighth intercostal spaces; if on the right side, the portion of the wall immediately above the liver and to the right of the sternum.

The possibility of detecting the exudate does not in any sense depend on its size, as the degree of dulness is dependent largely on the power of the thoracic wall to transmit vibrations, the amount of subcutaneous fat, the thickness of the muscles and skin, the expansibility of the lungs, the nature of the exudate, and the condition of the costal pleura. Just as in pneumothorax, a very large exudate may, on account of the amount of space within the pleural cavity, escape our notice, so in conditions of hyperresonance in the lung, and when the ribs are more ossified than usual, the effect of relatively large masses of exudate on the percutory note may be destroyed or at least diminished.

Dulness is most distinctly present in women and children who have relatively thin chest-walls. As a rule, from 400 to 500 c.c. can always be detected even under unfavorable circumstances, and much smaller amounts may under favorable conditions, as we have frequently proved by exploratory puncture, give rise to a considerable degree of dulness.

There is no doubt that much depends on the depth of the fluid, the amount of cells and fibrin contained, the swelling of the pleura itself, and the atelectasis of the superficial portions of the lung. I have seen cases of pleurisy without fluid exudate in which the necropsy showed that the solid deposits were neither very thick nor very extensive, and yet there was such a marked degree of dulness that if the results of percussion alone had been considered one must have infallibly assumed the presence of a tumor, particularly as in such cases the respiratory murmur is much diminished or altogether absent. And as in other cases, particularly in tuberculous plastic pleurisy with little or no involvement of the lung, the extent and intensity of the dulness over the upper and lower portions of the lung are often quite considerable and out of all proportion to the small amount of fluid exudate shown to be present by exploratory puncture, one is justified in concluding that the *intensity of the dulness* in general depends less on the amount of the exudate present or on a diminution of the air contained in the lung than on the sound-conducting power of the thoracic wall itself. In other words, large masses of exudate produce a relatively small amount of dulness if the chest-wall itself is capable of giving out a specially loud note; and, conversely, a slight variation in the intrapleural conditions or amount of air contained in the lung materially affects the note if the chest-wall itself does not contribute to the resonance.

The **method employed** in percussion must, of course, be varied according to circumstances, and the difficulties or uncertainties in the results obtained are frequently due to the fact that this circumstance is not sufficiently appreciated. If the thoracic wall is thick, deep percussion should be used; while if it is thin and the lung resonant, the examiner should percuss lightly, especially when determining the boundaries of the different areas of dulness.

It is an important rule never to try the percussion note of the diseased side until the conditions over the sound lung, its resonance in the resting position and in the various phases of respiration, the degree of compensatory activity or retraction, have been satisfactorily determined by means of percussion and auscultation and by carefully outlining the liver and stomach areas.

The louder the note over the sound lung or the apparently resonant portion, the less force should be used in percussion, remembering that under such circumstances even a moderate degree of dulness deserves consideration. Whenever the dulness is not very extensive, and particularly when it is desired to determine small areas or moderate degrees of dulness, light percussion should generally be employed, so as to avoid throwing the adjoining healthy portions of the lung into vibration.

The relation between the level of the exudate and its extent in the transverse direction must be taken into account. An exudate may under certain circumstances extend to a high level and produce a broad zone of dulness, although the actual amount of exudate is only moderate. Whenever retraction of the lung is interfered with by pulmonary emphysema, adhesions, etc., the thickness of the layer is naturally diminished, and the exudate accordingly rises to a higher level. As the layer of fluid is thin the dulness is less extensive, and although the exudate reaches to a high level, the three zones of dulness are not pronounced, the middle zone being absent or reduced to a minimum.

The same rule that has just been given for the determination of the tactile fremitus applies to percussion. The examination should only be made after the patient has taken several long breaths or coughed repeatedly, or else the difference between the results obtained immediately after the patient has assumed the sitting posture and after he has been breathing deeply for some time should alone be utilized in determining the distensibility of the lung and movability of the exudate.

The *upper limit of the exudate,* therefore, can be determined with certainty only after the patient has sat up for some time; and if in such cases no marked changes are observed after a certain time, it may be inferred that the exudate is quite large, or that the lung is much retracted, or that deposits and adhesions of relatively large extent and thickness have been formed. The upper limit of the exudate is indicated by a horizontal line if the patient is still capable of some movement, and particularly in patients who have continued to follow their usual work after the formation of the exudate. In patients who have been in the semirecumbent position for some time, it corresponds to a curve running from the vertebral column outward and downward toward the lateral wall, the direction roughly corresponding to that of the intercostal spaces.

In some rather obscure cases the upper concavity of the line is directed toward the sternum; in others the boundary-line is convex toward the sternum, and this form, in which the upper boundary-line corresponds to a parabola with its apex in the lateral wall of the thorax (usually in the axillary line), has been named after Ellis-Damoiseau.[9]

The formation of the curve depends not only on hydrostatic laws and the capillarity of the pleural space, but also on the character of the respiratory movements, the nature of the exudate, the formation of adhesions or other processes in the pulmonary tissue (atelectasis, bronchopneumonic foci, emphysema), and to some extent also on the condition of the chest muscles, the strength of the diaphragm, and other factors.

In regard to the diagnostic and prognostic significance of these curves, we possess no information, at least so far as my knowledge goes. According to Gerhardt,[1] a sigmoid curve with its highest point between the scapula and the axillary line is observed principally in individuals who are in the habit of lying on the diseased side, and is never found in recent cases.

On the *posterior surface* of the thorax two or three zones can be distinguished, as was shown on page 854 in speaking of the examination of the fremitus: a lower zone of flatness, and an upper zone where the note is normal, or somewhat louder than normal. These two zones are usually separated by a more or less extensive area where the note is loud or moderately tympanitic, corresponding to the retracted and relaxed, although not atelectatic, portions of the lung.

The higher the level of the fluid, the more these zones are displaced upward. With very large exudates the entire posterior surface may be absolutely dull, with the exception of an area near the vertebral column in the region of the interscapular space, where the retracted lung is closely applied to the thoracic wall. Here the sound may for some time remain moderately tympanitic. In certain cases the note obtained on deep percussion over the lower portions of the left lung assumes a tympanitic quality owing to the gastric and intestinal walls taking part in the vibrations.

In the **lateral wall** two zones only are usually observed: a lower one, where the dulness is moderate, and a smaller upper one in the axilla, where the note is moderately tympanitic. As soon as the exudate reaches the intrascapular space the last-mentioned zone also becomes dull and loses its tympanitic quality. In the anterior wall a low-pitched, loud, more or less tympanitic note is obtained even with a small amount of exudate, immediately beneath the clavicle and in the second intercostal space. If the exudate increases and it is possible to detect the presence of a fluid even in the upright position, percussion from below upward may show a gradual transition from absolute dulness to resonance or relative tympany, and even with a large exudate a clear tympanitic note is usually obtained close to the upper portion of the left border of the sternum.

A tympanitic note high, seldom low in pitch, is heard more and more distinctly as the atelectasis of the lung increases ; and even when the entire thoracic cavity is filled with fluid, a tympanitic element may be detected in the note immediately below the clavicle (Skodaic resonance) if it is possible to throw the column of air in the left bronchus and in the trachea into vibration. The note thus obtained is altered in pitch as the mouth is opened and closed (William's tracheal tone), and a cracked-pot sound is often produced, a circumstance which occasionally leads to errors in diagnosis (cavities) if the history is carelessly taken. It is readily explained if the physical conditions in the retracted lung are borne in mind.

During respiration the note varies even over the areas of greatest dulness. It becomes louder during inspiration because even the retracted portions of the lung become periodically filled with air, or because on deep percussion the actively expanding compensatory portions of the lung are thrown into more vigorous vibrations.

The loud, low-pitched tympanitic note below the clavicle (Skodaic resonance) becomes higher in pitch during inspiration, not so much on account of the inspiratory expansion of the pulmonary tissue as on account of the stretching of the intercostal muscles (Rosenbach[1]).

The percussion should include an examination of **Traube's space,** a semilunar space characterized by a loud tympanitic note bounded below by the left border of the thorax, above by a curved line with the concavity directed downward, and extending from the fifth or sixth costal cartilage along the border of the thorax to the anterior border of the ninth or tenth ribs.

In this region a narrow band of intense dulness may under favorable conditions be detected even when the exudate, as shown by exploratory puncture, is very moderate in amount. This dulness is readily distinguished from the splenic dulness because the transverse diameter of the area between the two axillary lines is relatively small in proportion to the vertical diameter in the mid-axillary line. If the dulness were splenic in character, the splenic tumor would in such cases be readily palpable below the costal border, hence the formation of an exudate in this region can be determined with certainty even without at once resorting to exploratory puncture.

The low-pitched tympanitic area known as Traube's space diminishes at each inspiration, and the note becomes higher in pitch the greater the distensibility of the lung and the downward excursion of the diaphragm. In the same way an accumulation of fluid in the lowest portions of the pleural cavity will tend to shorten it.

In the former case the presence of a loud non-tympanitic pulmonary zone furnishes a means of determining the magnitude of the diaphragmatic excursions. In the second case the detection of a more or less pronounced dulness within Traube's space is of some value in favor of the presence of fluid. The sign is not always quite conclusive, however, as other processes, such as acute infiltrations, abnormal distention of the abdominal organs with food, tumors developing in the hypochondrium, may bring about similar changes in the semilunar space, while, on the other hand, even large exudates may, instead of reducing the semilunar space, distend the thoracic wall or bring about contraction of the lungs.

Another difficulty is that unless the patient has been examined before the exudate is fully formed, or has been under observation before the beginning of a fluid exudate, the true extent of the semilunar space, which is subject to considerable individual variations, is not known, and the examiner therefore has no standard by which to determine whether the exudate is constant or has increased.

In extensive exudates the **diaphragm** may be so displaced, or, to speak more precisely, it may relax to such an extent from loss of tonus, that the semilunar space disappears completely and the entire lower portion of the thorax shows absolute dulness. Occasionally the dulness even extends below the costal margin.

Displacement of the liver is detected much more readily by palpation than by percussion, as the outline of the lower border of the liver and the extent of the respiratory excursions can be determined with much more certainty by the former method. While displacement of the liver is frequent, the corresponding condition in the spleen is rare unless the organ has become much enlarged by venous stasis or changes in the interstitial tissue.

Displacement of the heart has already been adverted to. There is one very important sign deserving mention, which consists in an *apparent enlargement of the cardiac dulness* in the transverse diameter sometimes observed in right-sided exudates. The enlargement, which may amount to 2 or 3 centimeters, usually presents a convex outline with the convexity toward the right, and merges into the hepatic dulness. It would be a great mistake to regard this phenomenon, which is often demonstrable quite early in right-sided effusions, as a certain sign of cardiac insufficiency or dilatation of the right ventricle, particularly as it is usually associated with a very moderate degree of dyspnea, a very good pulse, and vigorous cardiac action. A correct interpretation of this enlargement of the cardiac dulness, which usually begins immediately below the third rib, depends on a proper consideration of its peculiar outline, the intensity of the dulness, and particularly the absence of a distinct cardiac impulse, which, whether the heart were weak or strong, would necessarily show itself in marked heaving of the thoracic wall to the right of the sternum. It is evident, therefore, that the increase in the dulness is not due to displacement or enlargement of the heart; indeed, a careful examination will show that we have to deal solely with the effects of a pleural exudate. In a number of cases I have been able to prove, partly by direct exploratory puncture and partly by the subsequent course of the disease, that the dulness was not due to the heart, but to an early collection of exudate in that portion of the right pleural sac which covers the heart. This condition, it is true, is associated with a very slight accumulation of fluid in the lowest portions of the pleural sac below the ninth or tenth rib, as can be shown by careful light percussion. In the right lateral wall, on the other hand, the accumulation under such conditions usually occurs much later. Just as the appearance of this dulness is an early sign of right-sided effusion, so the disappearance of the dulness is a relatively early sign of beginning absorption. The value of percussion in determining the **resorption of an exudate** is comparatively slight. As a rule, it merely serves to confirm the results obtained by auscultation, except in cases of very extensive exudates, where it may indicate marked differences in resorption. If, however, a noticeable diminution in the intensity and extent of the dulness is coupled with an undeniable improvement in the respiratory murmur, if an indistinct or vesicular murmur is heard instead of no murmur at all, or one of the various forms of bronchial respiration, resorption may be assumed to have commenced. On the other hand, percussion is of value in determining the distensibility of the peripheral portions of the lungs, particularly if, even on light percussion, a considerable increase in the resonance is observed at each inspiration.

For the diagnosis of so-called *rétrécissement thoracique,* which is accompanied by the formation of massive plaques, percussion is of very doubtful value. Under such circumstances the results of a correctly performed *palpatory exploratory puncture* are far more reliable, as the crunching sensation and the peculiar resistance felt as the needle perforates the plaque are two of the most characteristic signs, not to mention that the failure to obtain fluid exudate absolutely confirms the diagnosis of commencing organization (Rosenbach[9]).

Alterations in the position of pleural exudates (more correctly, the movability of fluid exudates following a change of position on the part of the patient) have been specially studied by Gerhardt,[6] Rosenbach,[6] Strauch,[6] Nicolai[6], and others. With the method recommended by Da Costa, consisting in determining the limit of dulness while the patient is in a sitting posture, and again when he is lying prone, Strauch observed an alteration in the position of the exudate in only one case. Nicolai, on the other hand, found that it was distinctly movable in 55% of the cases, slightly movable in 30%, and failed to show any change of position in 15%. In my opinion, the question of the movability of a pleuritic exudate is so complicated that it cannot be decided by any one method of examination. When it is remembered what a marked influence the mere unfolding of the lung in deep inspiration exerts on the intensity and extent of the dulness, without the exudate necessarily undergoing any change of position, a mere increase in intensity—that is, an increase in the volume of the percutory note—cannot always be attributed to a displacement of the exudate. Since, moreover, the portions of the lung and thorax that undergo the greatest distention in the sitting posture are not the same as those which are most expanded in the dorsal or ventral position, the posterior portions containing more air in the latter posture, it is clear that, even without any change in the level of the exudate, an alteration of the percutory note may, and indeed must, be produced by any local alterations in the amount of air contained in the lung.

That the position of the exudate is due to the peculiar conditions of pressure within the pleura and is not always dependent on gravity alone, and that we have to deal with complicated static and dynamic conditions (the effect of aspiration, the effect of capillary spaces, local differences in pressure, etc.), is shown by certain observations made by Symington[6] on frozen cadavers containing pleuritic exudates of moderate extent. This observer found that in tranverse sections the anterior and lateral surfaces of the lungs were free from exudate, while the posterior surface was entirely covered by a layer of exudate of uniform thickness throughout its extent; and he also found that the inferior posterior complementary spaces of the lungs were entirely free from fluid and that the layers of the pleura were in contact.

Although the conditions under which these experiments were performed were so peculiar that their results cannot be accepted as conclusive in a clinical sense, they are not without some value in explaining the mechanism involved in the movability of pleural exudates. They at least support the view that the conditions on which the distribution of the exudate depends have not as yet been worked out in every direction, and tend to show that the outline of the dulness is not referable simply to processes which depend on the action of gravity.

[**Crepitation at the base of the lung** is said to be a sign of the presence of a very small effusion, so small as to be practically undiscoverable by the usual methods of examination. The crepitation differs from that heard in pneumonia, either in the beginning of consolidation or in the early stages of resolution; it is only slightly moist in character, more superficial and finer. The râles of resolution are more varied in size and much more moist. The sound under discussion can be differentiated from the râle of pulmonary edema, with which it may be associated. It is caused by pressure of the fluid and the relief afforded during inspiration, producing alternate adhesion and separation of the walls of the alveoli.*—Ed.]

The circumstance already referred to as the result of our own observations, that a displacement of the dulness is only observed after the patient has remained in the altered position for some time, is confirmed by Gerhardt,[1] and may possibly be owing to the presence of slight adhesions which must first be broken up. In his opinion, the view advanced by Laennec, that a displacement of larger exudates necessitates the movement of air in the individual portions of the lung, some areas in immediate contact with the exudate becoming atelectatic while others become expanded, is correct.

Gerhardt[1] also confirms what was found by Damoiseau,[9] that with a moderate exudate the sound side also becomes much dilated, and that the diaphragm on the sound side may even occupy a lower position than normal (see page 837). After puncture or absorption the diaphragm rises considerably on both sides, and a corresponding increase in the circumference of the chest is also observed on both sides.

This, as has already been mentioned, cannot be attributed to a passive dilatation of the healthy thoracic cavity by the thoracic organs being crowded to one side by the exudate, but rather to an active process depending on increased compensatory respiration.

4. AUSCULTATION.

The results of auscultation are most important at the beginning of the disease. With a little care the slightest roughening or deviation from the normal in the layers of the pleura can be detected by means of the characteristic rub resembling the creaking of leather. Auscultation may also enable the examiner to detect small masses of fluid exudate, particularly if a diminution in the intensity of the normal respiratory murmur is heard over a circumscribed area of the lower portion of the thorax, where the murmur is usually quite marked in men and children.

The **friction sound** is not always loud or creaking in character. Sometimes it is a soft rubbing sound or a short crepitation, which, however, presents certain differences from the typical crepitant râles that are often heard in pleurisy; the latter are specially noticeable over the posterior upper boundary of the exudate a few seconds after the patient has sat up.

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* W. Janowski, in *Zeitschr. für klin. Med.*, vol. xxxvi, Nos. 1 and 2.

The leather-creaking (*bruit de cuir neuf*) may be produced by other than pleuritic processes that are not due to inflammation, the eruption of miliary or larger tubercles, the presence of tumors, etc.; and even when there is a thin layer of fluid a distinct rub may be heard, as is easily shown by exploratory puncture.

A frequent source of error is a certain *symmetric and often palpable creaking,* elicited most frequently over the lower portions of the lateral wall, also over the suprascapular fossa or in the interscapular space, and rarely in the supraclavicular fossa. This phenomenon, which I have termed *pseudopleural friction,* so closely resembles the true pleuritic rub that even a practised examiner is not always able, without comparing the sound with the auscultatory note in other portions, to distinguish it from the creaking leather sound of the pleura. It appears to occur particularly in individuals with wide intercostal spaces, and when the excursion of the lower border of the lungs is extensive. This creaking, which is the best example of the so-called new-leather sound (*bruit de cuir neuf*), in by far the greatest number of cases is not only audible, but also, as has been mentioned, readily palpable, and possesses a certain jerky quality which is also characteristic of the pleural rub. In many cases where a soft rubbing sound may be detected by palpation, the acoustic impression received is not sufficiently characteristic to cause confusion of this fremitus which is determined by palpation with true pleuritic creaking. The phenomenon under discussion is a *muscular sound,* being produced by contraction of the intercostal thoracic and dorsal muscles, the intensity of which depends on the power of the muscle to which it is due and to the strength of the contraction and the sound-conducting power of the muscle. These factors are all present in a high degree when the intercostal spaces are wide and the subcutaneous cellular tissue contains little fat, conditions which obtain most frequently in the lower portion of the lateral wall of the thorax and in the interscapular space. In most cases confusion with the pleuritic rub is avoided by noting the symmetric character of the phenomenon so characteristic of muscular sounds and the remarkable constancy with which the sound is heard during a prolonged examination, the pleuritic friction sound, as is well known, altering its character very frequently. Finally, the diagnosis is rendered somewhat more easy by the fact that the sound is best heard during inspiration and may be entirely absent during expiration.

Pleuro-pericardial (pseudo-pericardial) **phenomena** possess a special significance. They are due to deposits in the immediate neighborhood of the heart, and are accordingly influenced to an equal degree by the cardiac action and respiration. They may even be affected more by the cardiac action than by changes in the respiratory phases.

The sound appears to occur most frequently in the region of the apex, where the heart is covered by a tongue-shaped portion of the lung. Its character is extremely variable; it may be systolic or diastolic, inspiratory or expiratory, and owes its existence to a roughening of the outer surface of the pericardium or the segments of the pleura contiguous with the heart. The acoustic phenomena vary according as the roughened surfaces rub against each other during the locomotion or contraction of the heart or only during the respiratory movements.

To determine whether the sound is produced within the pericardium, a protracted examination is usually required, and a decision is, as a rule, possible only if the patient can hold his breath long enough to eliminate completely the respiratory element of the sound. In certain conditions change of position, holding the breath, or coughing influences the character of the sound or causes it to disappear completely; and if this takes place, it is a positive sign that the phenomenon is extra-pericardial. The terms pneumo-pericardial, extra-pericardial, and pleuro-pericardial have all been used to designate the friction sound referred to.

The **changes in the respiratory murmur** in pleural effusions can be readily deduced from our knowledge of the possible changes in the local conditions. The intensity and character of the respiratory murmur depend on: (1) The possibility of acoustically effective waves being produced in the respiratory apparatus; that is to say, the character of the sound-producing medium. (2) The depth of the breathing. (3) The conducting power of the media intervening between the lung and the examiner's ear; that is, the faculty of the thoracic wall to transmit sound-waves, or, to speak more correctly, of being thrown into the proper form of vibrations.

The characteristic changes in the respiratory murmur in pleural exudates vary according as the conduction of the sound-waves to the ear of the examiner, which presupposes a synchronous vibration of the thoracic walls, is entirely suspended by the foreign medium, be it a layer of fluid or a solid exudate, or merely inhibited over a circumscribed area, or, finally, only certain forms of waves are transmitted on account of the abnormal conditions.

In the second place, the intensity and character of the respiratory sounds are influenced by *changes in the lung tissue,* which in turn depend on the extent of the exudate, for any change in the respiratory distention in the superficial or even in the deeper portions of the lung necessarily modifies the waves which produce the bronchial or vesicular murmur.

In moderately extensive and very extensive exudates the three factors mentioned are usually equally active, and hence the various forms of respiratory sounds (zones) are found developed most characteristically in such conditions. The intensity of the sound is found to vary in different areas up to complete disappearance of the vesicular murmur. Along with mere intensification and transition to the bronchial variety we find the most pronounced type of bronchial breathing.

Severe pain in the chest-wall restricts the respiratory movement and is to be regarded as a mechanical obstacle to the respiratory excursion, hence it necessarily weakens the intensity of the sounds or even causes them to disappear; but as under such conditions the intensity of the sound is diminished equally over all portions of the chest, this general modification of the respiratory phenomena always points to some general external cause, and not to a local disturbance of the tissues, whereas changes which produce a localized bronchial breathing always depend on some local tissue disturbance.

Diminution of the respiratory murmur may, of course, be combined with crepitant, or with non-resonant râles, or with sibilant and sonorous râles, if the retracted portion of the lung is still capable of distention during deep inspiration, or if, at least, during an excessive respiratory effort the air is able to pass the swollen bronchial mucous membrane. In the same way bronchial breathing may be associated with resonant râles produced in the larger bronchi. [Pitres made a series of experiments on human cadavers, injecting into the pleural cavity fluid that solidifies on cooling. His results tally in the main with the findings by physical examination. Small, vertical effusions occupy the groove between the diaphragm and the ribs in the axillary region and have a horizontal upper border. In the supine position the fluid sinks to the groove between the ribs and the vertebræ. In large effusions the upper limit of the fluid, in the erect posture, turns downward abruptly near the vertebral column, probably on account of the greater difficulty of displacing the lungs in that region. The form of these experimental effusions was modified by pneumothorax, adhesions, thickenings of the lungs, and increased resistance of the diaphragm. The lower limit was concave in small effusions, convex in large ones.*—Ed.]

As the exudate grows the respiratory murmur becomes more and more faint and distant, and finally disappears altogether. If the conditions are such that the production and transmission of the rough, sharp bronchial sound produced in the larynx or bronchi is still possible after vesicular breathing has been abolished by the retraction of the lung,—a condition which is always present in serous, slowly increasing exudates as one portion of the lung is overdistended with air and the fluid medium readily transmits sound-waves,—if, in other words, there is sufficient movement of air in the uncompressed portions, or perhaps even in the bronchi of the compressed portion, then the various modifications of bronchial breathing will of course be heard. The intensity and quality will, of course, depend on the site of its production and the conducting facilities; in certain cases it may have a distinct amphoric quality which occurs when the sound-waves from a large bronchus are conducted through a moderate extent of retracted lung tissue, and not only suffer no diminution, but even acquire a certain resonance.

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* From "Year-Book of Medicine," 1899.

The greater the difference between the bronchial breathing heard over the thoracic wall and the true laryngeal or tracheal breathing, a difference which must always be carefully determined by percussion, the more favorable are the conditions for the movement of air in the corresponding lung, and the less pronounced, as a rule, the dyspnea.

Accordingly, we not infrequently meet with cases, even in purulent exudates of children, where, instead of a marked degree of dulness with a slight tympanitic element, the bronchial breathing and fremitus may be as great as in pneumonia. In such cases it is to be assumed that we have to deal with a homogeneous fluid, that the pleura is not covered with deposits, that there is no edema of the thoracic wall, that there is no obstruction of the larger bronchi, and that the lung, though strongly retracted, is not compressed and has maintained the equilibrium characteristic of living tissue or is even in a condition of marked tonic excitation.

In very large exudates the thorax may be divided into *three distinct zones* of auscultation: a lower zone, where the respiratory murmur is entirely absent (compression or complete retraction of the tissue with diminished conduction); a central zone, where conduction is fairly well preserved, and more or less pronounced bronchial breathing is heard (moderate atelectasis); and an upper zone, where distinct and often increased vesicular breathing is everywhere audible. Between the shoulders and in the axillary space the bronchial breathing is often most marked, and in the lateral wall it is usually more pronounced than on the dorsal surface. As the level of the exudates rises, the lowest zone gradually increases in width, and the lower limit of bronchial breathing continually rises until the central zone may reach to the infraclavicular fossa, the respiratory murmur over the supraclavicular and supraspinous fossæ becoming indistinct. Eventually the respiratory murmur may disappear over the entire half of the thorax and only a faint remnant of sound be heard over the apices. In many cases amphoric breathing and faint metallic tinkling are heard in certain areas (in the interscapular space to the right and in the infraclavicular fossa) as soon as the exudate completely fills the pleural cavity. The lung, as a rule, then becomes permanently atelectatic and retracted upward to such an extent that the sound produced in the main bronchus becomes audible. In such cases we also obtain, as has been mentioned, a percutory phenomenon known as Williams' tracheal tone; in short, we have the phenomena erroneously regarded as pathognomonic of cavities.

With the progress of **atelectasis** and **hepatization** of the lung, which proceed hand in hand with diminution of the respiratory displacement and distention of the organ, the character of the respiratory murmur varies more and more in the different zones and at different times. Coughing and the expectoration of large masses of secretion may also modify the results of auscultation from one moment to another. Râles appear and disappear, vesicular breathing is replaced by crepitant râles or by bronchial breathing, or the inverse of this may, and indeed does, much more frequently take place.

As the physical conditions necessary for the production of so-called **bronchophony** are the same as those required for the reproduction of loud and high-pitched bronchial breathing, and only necessitate a relatively vigorous phonation, bronchophony will be heard more or less distinctly wherever bronchial breathing shows that the bronchi are clear, that retraction is moderate, and the conducting power fairly good. In such cases also the modification of bronchophony which, on account of the high-pitched jerky bleating quality depending on a peculiar periodic augmentation and interruption of the sound-waves, is termed **egophony,** and which formerly played an important rôle in the diagnosis of pleuritic exudates, is most apt to be observed.

The so-called *phenomenon of Bacelli,* consisting in increased conduction of whispered sounds, is not, as Bacelli[6] assumed, characteristic of serous exudates, for it can be shown by exploratory puncture that the phenomenon may be present with equal distinctness in croupous pneumonia, and even in chronic infiltration of the lung, whereas it is absent in simple serous exudates and in hydrothorax, even when the lung tissue is healthy.

In addition, a number of other factors, such as complication of pleural effusion with atelectasis or bronchopneumonia, the strength of the patient and his ability to speak in a loud tone, the thickness and condition of the thoracic walls, may materially modify the effect of the exudate itself on the conduction of the sound-waves, for all of which reasons Bacelli's phenomenon, or the conduction of whispered sounds in the back of the thorax, is dependent on precisely the same conditions as bronchial breathing.

The faintest vibrations of the air column in the bronchi are transmitted to the thoracic wall without any diminution of intensity even when there is a certain degree of atelectasis in the parenchyma, if the air-passages are not obstructed or compressed, and the lung, on account of its relaxed condition, furnishes a good medium for the conduction of the sound-waves; hence whenever loud bronchial breathing is heard in the back, the whispered voice will also be distinctly audible (O. Rosenbach[6]).

Auscultation of the heart also may afford valuable information in regard to the condition of a pleural exudate. If the heart sounds are heard more distinctly than usual over an area of dulness, it betokens an infiltration. At least, it may be accepted as a fact that exudates encapsulated near the anterior surface of the thorax are more apt to produce a diminution than an augmentation of the heart sounds. If infiltration of the lungs is excluded and the heart sounds are very loud outside the limits of the normal heart area, dislocation due to the presence of an exudate may be assumed if this is confirmed by the results of percussion.

Heart murmurs also may be produced by an exudate. In the presence of the usual signs of displacement of the heart a murmur heard over the large vessels points to commencing compression or obstruction to the blood-current, while a diminution of already existing venous murmurs almost always indicates an increase in the positive intrathoracic pressure, since the intensity of the venous hum is almost proportional to the rapidity of the venous flow toward the thorax.

It goes without saying that in any pleuritic disease the continuance of a high temperature may lead to the production of accidental murmurs due either to a febrile acceleration of the blood-current or to great impairment of the heart-muscle, although they are not in any sense characteristic of a pleural effusion. As far as my observation goes, murmurs of this kind can never with certainty be referred to compression of the aorta or of the vena cava. Such an event is, however, quite possible on theoretic grounds.

5. EXPLORATORY PUNCTURE.

Not without reason much importance is not attached to exploratory puncture in the diagnosis of pleural effusions. Not so very long ago it was often a very difficult diagnostic problem to determine in doubtful cases whether there was or was not an effusion in the pleural cavity, or an inflammatory affection of the lung associated with diminution in the amount of air, or a complication of both processes ; and to determine the nature of the effusion, whether purulent, serous, putrid, or bloody, demanded the most subtle reasoning. Now, however, thanks to the simple and universally employed method of puncture with Pravaz's syringe, any doubt on these points can be removed with certainty and without danger.

An early recognition of the nature of the fluid is particularly important in purulent and putrid exudates, as surgical intervention (incision or permanent aspiration) in such cases affords the only successful means of treatment, and the chances for complete recovery are more favorable, the earlier the radical operation is performed.

Whenever, therefore, a positive diagnosis is not given by the existing symptoms, which is usually the case at the beginning of the disease, exploratory puncture should be resorted to in order to determine the nature of the condition. It is, as I said fifteen years ago, an *independent diagnostic method* which in many cases not only completes the usual physical examination by means of percussion and auscultation, but also places in our hands a means of measuring the value and reliability of those methods, and it is only by a systematic employment of exploratory puncture in doubtful cases, the number of which increases more and more as soon as we begin to doubt the reliability of the usual signs, that we reach the conviction that our other diagnostic methods, valuable though they be, are not as trustworthy as might be desired.

The usual objection raised to this method, the diagnostic value of which cannot be overestimated, is that a negative result is not an indisputable sign of the absence of fluid because, owing to the minute caliber of the needle used, the resistance of friction is not overcome by aspiration, and, therefore, though the purulent focus may be reached by the needle, the pus, if it is thick or contains small coagula, is not always drawn into the cylinder of the syringe.

That there is some truth in this objection will not be denied by any one who has performed any number of exploratory punctures, especially in deep-seated suppurations,—perinephritis, pelvoperitonitis, perityphlitis, etc.,—in which operation and ultimate cure in most cases depend on early diagnosis. In these very conditions, where a long needle is needed and the pus is apt to be thick, aspiration through the comparatively narrow caliber of the needle becomes very difficult, as the resistance offered by friction is of course much greater in a longer tube. Hence it happens much more often than in exploratory puncture of the thoracic cavity that only a few drops of a purulent fluid are slowly drawn into the cylinder of the syringe. In fact, the syringe not infrequently remains completely empty or the piston even springs back when it is released, and the examiner either decides that there is no pus present or, if he wishes to make sure, has to repeat the puncture in other places, possibly with the same want of success. But after a great deal of experience I have learned that an apparently negative result cannot really be regarded as negative unless the needle itself is found to be entirely empty and does not contain the smallest drop or slightest shred of pus, because it often happens in consequence of an unusually high resistance that the pus does not penetrate beyond the very point of the needle. It is especially apt to happen if at the beginning of aspiration a minute shred enters the needle and by adhering to its walls encroaches on the lumen or obstructs it altogether.

To allow for this possibility I find by experience that it is wise, in any case where after raising the piston one-third no fluid enters the cylinder, to withdraw the syringe at once without allowing the piston to slide down to the end of the barrel. If the piston is allowed to slip back by the pressure of the air, or is pushed back while withdrawing the syringe, not only would any fluid contained in the needle be returned to the interior of the body and the experiment remain doubtful, but the pus or other products of inflammation contained in the cannula might be deposited in healthy tissue, and thus allow the infection to spread. In puncturing the intestine or putrid cavities it is especially important, after the completion of aspiration, whether the result be negative or positive, to keep the piston firmly fixed with the fingers while the needle is withdrawn as rapidly as possible. After an apparently negative puncture of this kind has been completed, the examiner should at once satisfy himself as to the presence and nature of any fluid within the needle. This is best done by holding the tip over a cover-slip and carefully pushing down the piston. In this way it will not rarely be found that a small droplet of pure pus, recognized as such even macroscopically, or a minute purulent floccule floating in a drop of blood, was contained in the needle, and if necessary the finding can at once be verified with the microscope. By this procedure I have more than once been enabled to confirm a provisional diagnosis, which either induced me to advise an operation revealing an extensive purulent deposit, or at least inspired me with enough confidence to make another exploratory puncture at the same spot with a larger needle.

My advice, therefore, even when the result of exploratory puncture is apparently negative, is never to regard it as negative, that is to say, to exlcude the presence of a purulent effusion at the site of the puncture, unless the minute droplet of usually sanguineous fluid contained in the needle on microscopic examination distinctly shows the absence of larger numbers of pus-corpuscles than ought to be found in the amount of blood present.

Quite frequently a dulness in the posterior inferior portions of the lung, which apparently could be due to nothing but consolidation of the lung itself, is found on exploratory puncture to indicate a pleural effusion. In many cases of apparent atelectasis the same thing happens; and, conversely, the presence of a pleural effusion is often assumed where exploratory puncture shows that we have to deal with consolidation of a lung or with an old adhesion. The frequency of these errors can only be appreciated by systematically practising exploratory puncture as a third method equal in value (in most cases) to the ordinary physical methods; that is to say, it should be used not only in all distinctly doubtful cases, but in all cases that show the least variation from the typical picture. This is a perfectly justifiable procedure, since experience shows that it is quite harmless and yields prompt results.

Valuable information may also be obtained by a modification of this procedure which has not heretofore received much attention. It is the so-called *palpatory puncture*, "akidopeirastik," in the thoracic cavity.[9] With a little practice the examiner soon learns to judge of the condition of the pleura by the amount of resistance encountered by the entering cannula and the ease with which it can be moved to and fro in the thoracic cavity. If the resistance is abnormally great, and the passage of the cannula is accompanied by a perceptible crunching noise, it may be inferred with a reasonable degree of certainty that there are adhesions between the layers of the pleura, with atrophy or induration of the pleural tissue and cirrhotic changes in the lung. Since these pathologic alterations can in many cases be established beyond a doubt by a postmortem examination, this method enables us to recognize conditions better than any of the other accepted methods of examination.

With moderate care the sources of error which exist in this as well as in any other method can be reduced to a minimum. In the first place, the syringe must be well disinfected and the needle, which should be twice as large as the ordinary hypodermic needle, should be securely fastened to the syringe. With these precautions, if the skin is held firmly between the fingers so as to avoid striking a rib, and by taking into account the thickness and character of the thoracic walls in regard to edema, etc., so as not to penetrate too deeply and injure the lung,—an accident which must of course be carefully guarded against, although it is not at all serious,—reliable results will be obtained and there will be no danger of the needle being broken.

As soon as the needle has entered the thoracic cavity it should be ascertained whether it moves freely and, if necessary, the instrument must be pushed further in or partially withdrawn. The piston must be drawn back as slowly as possible, for, owing to the sudden diminution of the internal pressure, the outer air, being under higher pressure, might force itself into the cavity at the junction of the syringe and the needle, especially if the needle does not fit tightly on the syringe. But even if this should occur, a sufficient quantity of exudate is usually obtained for diagnostic purposes.

Even when the exudate is very small in amount, satisfactory results may be obtained if the needle is introduced within the area of dulness obtained by percussion; that is to say, above the lower border of dulness. In the special form of effusion characterized by the abundance of cellular elements which sink to the bottom and form a lower layer rich in morphologic constituents (purulent layer) underneath a clear serous layer, puncture at the lower border of the area of dulness at once reveals whether there is any tendency to suppuration.

If there is any suspicion that the exudate is purulent,—that is, when there is irregular fever that cannot be accounted for,—the examiner should not rest satisfied with a negative result obtained with the ordinary short needle of a Pravaz syringe, but should try one or more punctures in the area of dulness with a needle of double the length. By this procedure, which in the hands of a practised and careful physician is perfectly safe, the presence of a deep encapsulated exudate between the lobes of the lung may often be discovered (see page 893). [Care must be taken not to puncture if the skin is the seat of an infection. Under these circumstances a serous exudate may be infected by the entering needle, infected in its passage through the inflamed skin.—Ed.]

I cannot, therefore, agree with Fräntzel[9] that the value of exploratory puncture has been overestimated or that the sources of error in this method are more numerous than in other methods of diagnosis. The results I have obtained by this method have been uniformly good; but at the same time, I must not omit to point out that exploratory puncture, like all other methods of diagnosis, does not always yield direct pathognomonic results, although the information gained is more uniformly positive than that obtained by any other method.

I have never seen any harm come to the patient from the small wound occasioned by the puncture, not even in cases where the lung was touched, an accident that cannot always be avoided. I have repeatedly had the opportunity of convincing myself, both by experiments and at autopsies held shortly after the performance of an exploratory puncture, that the worst result that can happen is a small superficial ecchymosis which disappears within two days after puncture has been performed.

Whenever there is a *possibility of operating,* exploratory puncture ought to be resorted to very early, as it offers the most rational means of arriving at a clear understanding of the case. I can remember a number of cases where the clinical manifestations, except for a certain pallor and loss of strength, pointed merely to the existence of a moderate serous exudate, and where exploratory puncture revealed the presence of a thick purulent or, in some cases, even a putrid exudate. Since the prognosis in any operation undoubtedly depends on its being performed early, exploratory puncture may in such cases properly be termed a life-saving and health-preserving procedure.

Exploratory puncture may also be utilized in the *diagnosis of malignant tumors,* not so much by bringing out particles of the tumor or cells of a typical character, as by showing that the dulness is due to the presence of a solid body or that the exudate contains a large amount of pure blood. [Cells of a typical character have been removed with fluid drawn by exploratory puncture and a diagnosis of carcinoma made thereby. Steele and Girvin had such good fortune in diagnosis.—Ed.]

It also affords the most reliable means of determining whether a tumor in the breast-line only simulates fluctuation, as in soft enchondroma, sarcoma, multilocular echinococcus, or whether one has to deal with true fluctuation, as in accumulations of fluid within the pleura, in peripleuritic abscess, or the ordinary form of echinococcus.

The value of exploratory puncture for the *bacteriologic diagnosis* hardly needs to be mentioned; it is during life the only means of securing an opportunity to determine the nature of the micro-organisms contained in the exudate by means of the microscope or culture-tubes. This is not to be taken as an admission that any great value is attached to the bacteriologic diagnosis of pleural exudates, a question which has already been sufficiently treated in the sections on Etiology and Diagnosis.

The *importance of early exploratory puncture* is shown in the two following cases. It is seen by one of these cases that the fluid obtained by puncture must be carefully examined as to its odor, because the exudate may retain its serous character even after putrefaction has set in in the lung and pleura.

Case I.—*Putrid Pleurisy Presenting the Signs of a Moderately Large Serous Exudate. Exploratory Puncture. Operation. Recovery.*

A rather delicate and very anemic girl, twelve years of age, was suddenly taken ill and for a week presented the signs of a moderately large pleural exudate with a moderate degree of fever in the evening and a subnormal morning temperature, interrupted occasionally by days of complete freedom from fever.

The appetite from the very beginning was much diminished and there was marked loss of strength. Although neither the size of the exudate nor the character of the physical signs was such as to indicate anything but a serous exudate, an exploratory puncture was nevertheless decided on in view of the character of the fever curve and the evident impairment of nutrition. I had expected to find a serous or perhaps a hemorrhagic exudate, but to my great surprise I discovered that the exudate consisted of an extraordinarily fetid greenish-yellow, seropurulent fluid, which of course necessitated a radical operation without further delay.

At the operation considerable masses of offensive, somewhat hemorrhagic purulent material were evacuated, and within a few days a material improvement in the appetite and general condition took place. In spite of the apparent gravity of the case, the lung rapidly expanded, and after ten weeks the wound was completely healed and the normal function of the lung entirely restored. The patient was permanently cured.

Case II.—*Puerperal, Multilocular Pleurisy with a Serous Exudate which in Some of the Cavities was Fetid. Operation. Recovery.*

A puerpera, thirty years of age, developed symptoms of pulmonary infarct with secondary pleurisy. The attending physician made an exploratory puncture and found a serous exudate so that operative intervention appeared unnecessary, but as the patient within the next few days developed a very high temperature with occasional chills, puncture was repeated, and again a serous exudate was obtained, which, however, when examined by the sense of smell, was found to be exceedingly fetid.

A multilocular pleurisy was accordingly diagnosed and exploratory puncture repeated at another point, where pus was then obtained. Incision was then determined upon and a multilocular pleurisy, containing both serous and purulent, but uniformly putrid exudate discovered. Complete evacuation of the pleural cavity was achieved only after all the adhesions had been broken up by means of the index-finger and an elastic catheter introduced through the operative wound. This case also, which was treated with iodoform, ran a favorable course and terminated after twelve weeks in complete recovery, although the patient's strength was much impaired.

Another case, reported on page 895, in which there was an empyema encapsulated between the lobes of the lung, is also very instructive, both from a diagnostic point of view and as showing the importance of exploratory puncture. It also shows the importance of making a preliminary exploratory puncture at the point of incision before proceeding to operate.

[**Radioscopy.**—In certain conditions, as in encapsulated pleurisy or in the presence of emphysema, radioscopy may be of use in the diagnosis of pleurisy with effusion. Owing to the blurring of the picture by the movements of the heart, the chest-walls, and the diaphragm during respiration, radiographs are less satisfactory in the examination of the chest than the fluoroscopic screen. For a complete description of methods and equipment the reader is referred to "The Roentgen Rays in Medicine and Surgery," by Francis H. Williams. Cabot also devotes an appendix to the subject in his "Physical Diagnosis of Diseases of the Chest." In emphysematous subjects, and when there is marked compensatory hypertrophy of the sound lung, it is often difficult to determine the position of the heart by percussion, whereas these conditions render radioscopy easier. When a small effusion is present, the normal curve of the diaphragm appears flattened. Williams considers the method to be of no value in distinguishing between empyema and a serous effusion. Walsham, on the other hand, believes it is possible to distinguish between a serous effusion and empyema by the greater density of the shadow in the latter condition.—Ed.]

6. CLINICAL COURSE OF UNCOMPLICATED PLEURISY.

It is, of course, impossible to give a description of pleurisy applicable to all cases. Some are violent at the outset and yet end in recovery, while there are complicated cases which begin insidiously and almost imperceptibly and run on to fatal termination. As a rule, violent initial symptoms indicate not only an unusual irritability, but also an excessive degree of irritation. In such cases the exudate may be expected to accumulate again and again and to attain large proportions within a short time, while in cases having an insidious onset, unless one has to deal with a much debilitated individual, the exudate usually does not exceed a moderate amount.

The cases belonging to the first category, excluding cases of *pleuritis acutissima*, in which the prognosis is unfavorable, are usually characterized by violent onset and rapid recovery, whereas those in which the exudate accumulates slowly are apt to remain stationary for some time.

The presence of a friction sound over the entire thorax during the initial stage is not by any means the forerunner of a large and diffuse effusion, nor are such cases always due to a latent tuberculosis, since recovery often takes place without the formation of a fluid exudate. A friction sound over the lower portions of the chest in front is a comparatively rare initial symptom of extensive pleural effusions; hence if the friction sound is loud, a mild rather than a severe case may be expected, unless tuberculosis, pyemia, or carcinomatosis lies at the bottom of the trouble.

Many cases show a striking constancy in all the phenomena; others, again, are marked by the occurrence of frequent relapses with very high fever; in these the prognosis is usually very unfavorable.

It is comparatively rare for an acute pleurisy with rapid increase of the exudate and a rise of temperature above 41° C. (105.8° F.) to end fatally with delirium and acute heart failure. Death from exhaustion and increasing dyspnea is much more common. Not infrequently, absorption, after proceeding favorably for a while, is suddenly interrupted, and many cases fail to undergo absorption even after operative evacuation; the fever, which may be moderate with only slight evening elevations, as well as the physical signs and the dyspnea, persist. Such cases are usually due to tuberculosis, or else they eventually end in tuberculosis.

In *secondary* pleurisy, as defined on page 805, the ultimate result usually depends on the intensity and extent of the basal disease which is the primary etiologic factor. Pleurisy complicating rheumatism is more apt to end favorably than one depending on tuberculosis, although even here recovery is possible. Even in carcinomatous pleurisy temporary or partial absorption may take place with the subsidence of the purely inflammatory manifestations.

The ultimate outcome in *putrid* or *purulent* effusions, unless operative intervention is early resorted to, as now generally happens, depends on the possibility of rupture and the circumstances attending such an event. If the pleurisy is not merely a symptom of pyemia, or if general pyemia does not take place, recovery is possible in various ways. Even a long-protracted and extensive serous pleurisy may eventually end in complete resorption of the exudate and perfect recovery, so that from three to six months after the beginning of the disease not a trace remains to indicate the former condition. But in many cases diminished expansion of the lower portions of the thorax, a slight dulness on percussion, diminution of the fremitus or respiratory murmur, or the presence of some variety of râles indicate the existence of certain alterations in the tissues. The patient often retains a sense of constriction or feels a stabbing pain on violent exertion, although the interference with the respiratory mechanism may not be demonstrable by physical means.

In the later stages of pleurisy and many years after complete recovery, so far as the function of the organ is concerned, a peculiar sound resembling a moderately loud vesicular râle may often be heard over the lower portions of the thorax. The sound is constantly present; it may be continuous or heard only during inspiration or expiration and is not easy to interpret, as it may be equally audible during very shallow respiration.

After an examination of a number of cases, and from the fact that it is constant and occurs only after pleurisy with effusion, I am inclined to attribute the sound to a permanent abnormality in the layers of the pleura. It may also be a mere bronchial or pulmonary râle due to a partial atelectasis, to chronic bronchitis, or to the presence of minute bronchopneumonic foci. In favor of its being produced in the pleura we have, as above stated, the localization over the lowest portions of the dorsal surface, the intensity and uniformity of the sound, and the anamnesis; but in absence of any postmortem findings no definite conclusion can be arrived at.

Even *rupture of the exudate*, either external or internal, may be followed by complete recovery *quoad functionem.* But alterations in the external form of the thorax can be seen for some time afterward, either on the affected side alone or on both sides. There is in such cases more or less deformity of the thorax, a marked dislocation of the thoracic and abdominal organs, emphysema, and some interference with the respiratory movements.

The nature of the disease can thus often be determined many years after the occurrence of perforation, and numerous cases which, before the era of the tubercle bacillus, were regarded as phthisis on account of the severity of the symptoms, and found their way into the statistics as cases of recovery from phthisis, unmistakably show by the presence of certain characteristic signs that we have to deal with a case of complete recovery from empyema after rupture and not with pulmonary phthisis.

Before the days of exploratory puncture, when hectic sweats, loss of strength, paroxysmal attacks of coughing, remittent fever, and abundant sputum were wont to be regarded as signs of phthisis, diagnostic errors of this kind were common.

A purulent exudate may rupture through a bronchus, or through the thoracic wall (*empyema necessitatis*), or into other organs, into the abdominal cavity, and into the intestines. Encapsulation of pus between the liver and the diaphragm is not infrequent, and may be due to direct rupture or to extension of the inflammatory process by continuity. Even in these cases spontaneous cure, though rare, is possible. As a rule, however, unless the condition is recognized, the patient eventually dies after a period of continued fever with frequent chills, or with symptoms of diffuse peritonitis, gradual cachexia, and amyloid degeneration.

Now that exploratory puncture and timely operative intervention are the rule, and the practice of irrigating during the after-treatment has fallen into disuse, shrinking of the lung following a purulent exudate is almost never observed. In the case of serous exudates it still occurs in spite of operative intervention, but never attains a very high degree unless there is some grave disease of the lung tissue. The permanent effects are, as a rule, limited to a slight functional disturbance, a moderate contraction of the intercostal spaces, and fixation of the ribs and of some of the costal articulations.

Many cases end in *partial* or *total adhesion of the layers of the pleura* (obliteration of the pleura), which, strange to say, in some cases does not appear to occasion any great inconvenience, while in others it is apparently the only cause of very considerable disturbances. The determining factors in this respect still await explanation, so that the diagnosis and prognosis of pleural adhesion are exceedingly difficult. It appears, however, that the result depends less on the extent of the adhesions than on the thickness of the newly formed connective tissue and the degree of already existing emphysema or contraction of the lung. Obliteration of the complementary spaces appears to be followed by the worst consequences, as it interferes greatly with the movement of the diaphragm.

In addition to obliteration of the pleural sac, which more or less interferes with the efficiency of the circulatory apparatus, and in addition to dislocation of the heart and interference with its movements by plastic exudates on the outer surface of the pericardium, we also have morbid processes in the mediastinum resulting in compression and distortion of the larger vessels, or at least interference with the movement of their contents (brawny mediastinitis and mediastinopericarditis).

It is difficult to decide whether the hypertrophy and dilatation observed in total obliteration of the pleura are directly due to an adhesion, as there are cases of extensive chronic adhesion without any cardiac alterations. There is no doubt, however, that such a process in the pleura may in various ways produce circulatory disturbances within the heart, since the volumetric changes in the lung, which constitute one of the most important factors in the circulation, largely depend on the size of the complementary spaces in the pleura, not to speak of changes in the reflex innervation through the vagus produced by any irritation of the heart by the pleura.

Retraction of the lung is generally followed by more or less extensive bronchiectatic changes which sometimes produce very characteristic symptoms.

The formation of permanent internal or external *pulmonary fistulas* is of course very unfavorable; owing to the profuse suppuration they, as a rule, lead to general cachexia or grave renal diseases, either parenchymatous nephritis or amyloid degeneration, within a very short time.

Another unpleasant complication is *thickening and retraction of the covering of the liver* and obliterating *pericarditis;* I have seen the latter follow immediately upon a pleurisy or develop some time after its termination, and it is difficult to decide whether the pleurisy is the cause of the disease in the other serous membranes or whether one has to deal with a kind of fibrous diathesis.

These plastic exudates rarely undergo calcification. Calcification is more apt to take place when a permanent thoracic fistula results from an empyema on account of insufficient expansion of the lung.

In very rare cases *concretions* resembling pulmonary and bronchial calculi are met with in the pleura.

Both tubercular and non-tubercular disease of the pleura is often followed by *pulmonary* or *glandular tuberculosis* because the tissues of the insufficiently dilated organ afford a favorable soil for the development of structural disturbances within the parenchyma itself and the lymphatic apparatus. Tuberculosis of the pericardium is also occasionally met with.

General or circumscribed *emphysema* of the diseased or of the healthy lung (*volumen pulmonum auctum*) is a common result of imperfect recovery from pleuritic affections. I have seen a number of cases where a complete solidification of one of the lower lobes, which was covered by thick connective-tissue plates, existed side by side with intense emphysema of one of the other lobes, or of both lungs.

Caries of the ribs is comparatively rare even in cases of long-protracted purulent exudates, as necrosis and ulceration of the bony tissue is prevented by the ossification, which is usually quite extensive.

A complication which will be adverted to again in speaking of the progress of the disease after operative intervention consists in certain *congestive phenomena* which, under certain conditions that are not quite understood, declare themselves in the lung of the diseased or of the sound side, and occasionally go on to distinct *edema;* they may even end fatally, with the symptoms of acute or chronic pulmonary edema or sudden cardiac failure.

The *edema* which occurs either in the sound or in the diseased lung can in rare cases be explained on the theory advanced by Cohnheim and Welch and supported by the results of their experiments. The writer has seen a number of cases with moderate exudates in which the theory of antecedent weakening of the left ventricle with normal action of the right was quite inadmissible, as the symptoms of edema were not preceded or accompanied by diminution in the strength of the pulse or heart sounds such as would indicate a weakening of the left ventricle. It is at least quite certain that in the great majority of cases observed by the writer the accentuation of the second pulmonary sound, characteristic of increased activity of the right ventricle, was absent. Besides, edema may be accompanied by appearances of general cardiac weakness, that is to say, a diminution in the working capacity of both ventricles.

It is probable that edema usually develops only because of alterations in the work of the protoplasm itself, and is only secondarily produced by disturbances in the walls of the pulmonary vessels. Whenever the distention of the vessels produced by stasis or an excessively acute dilatation exceeds the power of the protoplasm to expedite the flow of blood, the serum escapes from the distended vessels and accumulates in the surrounding lymph-spaces in spite of the great increase in the local work performed.

Hence the so-called permeability of the vessel-wall is, in our opinion, synonymous with relative or absolute insufficiency of the protoplasm of the organ, as a result of which the chief part of the work is thrown upon the lymph-spaces and lymph-channels. This insufficiency is produced by any diminution in the efficiency of the expediting powers of the organ; in other words, its specific cells. Under normal conditions transportation is effected chiefly in the blood-vessels, and only to a moderate extent in the lymph-channels and lymph-spaces; but under abnormal conditions the greater part of the work is at least temporarily transferred to the intercellular spaces, and the excess of material, owing to the failure of the mechanical process of drainage, is not returned to the blood, but accumulates in the reserve spaces.

This is the only possible explanation of the sudden occurrence of edema in a lung which is expanding after the evacuation of an exudate, for the vessels in such cases are not always diseased, as is shown by the fact that the edema, although developing rapidly and at first assuming large proportions, very often disappears altogether in a short time, the function of the lung being completely restored. If there were really a profound lesion of the vessel-wall, such a rapid *restitutio in integrum* could not take place.

Accordingly, we have to do with a disturbance which is produced by relative functional insufficiency, a temporary inability of the tissues to expedite a comparatively large mass of blood which is compensated for without any detriment to the organism as soon as the relative engorgement has been diminished by drainage into the lymph-spaces.

We have every reason to believe that a lung which has for some time been retracted or even compressed receives, after the sudden evacuation of a large mass of exudate, much more than the amount of blood which was circulating during the period of compression, and this blood, of course, flows into the expanding lung as into the interior of a sponge, so that the resorptive power of the tissue and the facilities for drainage become inadequate.

A flooding of the uncompressed portion of the diseased lung, or even of the healthy lung, may of course take place, as after the evacuation of a very large exudate much more blood can find its way to the lung than under normal conditions, the amount varying with the excess of pressure within the thoracic space.

The extent to which the sudden removal of an abnormal pressure may affect the circulatory equilibrium is seen when, after the evacuation of an ascitic fluid, a large amount of blood is allowed to flow into the abdominal organs which have suddenly been released from pressure, an event which is not rarely followed by symptoms of cerebral anemia unless certain precautions to prevent a too sudden diminution in the pressure are observed.

We believe, therefore, that *acute edema of the lung in pleurisy*—be it that the fluid is derived from the bronchial arteries and is poured chiefly into the bronchi, or from the capillaries of the pulmonary artery and escapes directly into the alveoli—is in most cases to be regarded as the expression of an acute congestive hyperemia, and only in rare cases as the expression of a so-called stasis due to weakness of the left ventricle, or, in other words, as a venous hyperemia due to absolute insufficiency of the blood-vessel walls.

Sudden death is not a very rare occurrence. It is brought about either by heart failure, which shows itself chiefly in a sudden unexpected enlargement of the heart dulness to the right and symptoms of intense venous stasis, or by the sudden compression or occlusion of the aorta, or—and this is probably a more frequent cause—by obstruction of the vena cava at the point where it pierces the diaphragm. Death often results without any demonstrable anatomic changes in the organs, and is immediately preceded by convulsions like those chiefly observed in acute cardiac failure or cerebral anemia.

Death as the result of pulmonary embolism or thrombosis is comparatively rare. It occurs usually in patients presenting evident signs of cachexia or the phenomena of cardiac dilatation and cardiac or other thrombosis, especially *thrombosis in the distribution of the femoral vein.*

Leichtenstern[8] advances a theory, based on experimental investigation, that compression of the vena cava cannot be the true cause of death because the vein, which even after profound alterations remains permeable to water, is not actually doubled on itself by the pressure of the exudate, but merely crowded over to one side.

This view is emphatically contradicted by certain experiments performed by the author (page 840), and it is to be remembered that the conditions during life are somewhat different from those in the cadaver. Furthermore, the pressure of the exudate is not the direct cause of the kinking in the vena cava. The vein, after being displaced by the alterations within the thoracic cavity, is compressed by a sudden violent action of the descending diaphragm, or by some other cause, to such an extent that circulation becomes arrested.

In very rare cases *death from asphyxia* appears to be produced by a sudden flooding of the lung after *rupture* of a purulent exudate into the bronchi. It would seem that such an accident could occur only when rupture takes place during sleep, and in such cases it must be assumed that the fault lies in abnormal weakness of the heart or lungs, not to speak of the possibility that death may be due not only to the filling of the bronchi with pus, but also to simultaneous edema of the lung. The perforation is rarely so great as to permit large masses of exudate to escape at once and inundate the bronchi.

Rupture into the trachea, hemorrhages from ulcerated pulmonary pneumothorax, diffuse bronchitis, and cerebral embolism are occasional causes of sudden death.

Among the **sequels of pleurisy** are to be mentioned: General anemia, continuous or hectic fever gradually exhausting the patient without marked alterations in the physical signs and without signs of tuberculosis in the lungs; amyloid degeneration of all the organs (this disease occurs, as a rule, only after purulent pleurisy with caseation or fistula formation); chronic nephritis with general or local edema which may be due simply to a mechanical interference with the circulation, and accordingly not accompanied by inflammation or degeneration of the renal tissue; and, finally, tuberculosis, secondary suppurations, and retroperitoneal and intraperitoneal processes.

Thrombosis of the femoral veins (so-called phlegmasia alba dolens) is a not uncommon and ever to be dreaded sequel, as it not only retards convalescence, but, by condemning the patient to lie on his back for a long time, also favors the formation of atelectasis and hypostatic conditions in the already weakened lung, and, by the production of pulmonary embolism, often becomes the cause of sudden death.

X. COMPLICATIONS.

Many complications in pleurisy are observed after operation, either puncture or the radical operation, and will, therefore, be discussed later. For the present I should like to call attention to the following points.

According to what has been said of the general course of the disease, the most frequent complications consist of acute and chronic infiltration of the lung, thrombosis of the femoral vein, inflammations and degenerative atrophy of the thoracic muscles, contractures of the thoracic muscles, digestive disturbances, swelling of the liver, congestive phenomena in the extremities, distressing paroxysmal cough, insomnia, edema of the skin in places subject to constant pressure, especially on the back, bed-sores, and petechiæ in various portions of the body.

In chronic and particularly in tuberculous pleurisy a great variety of *cutaneous hemorrhages* are not infrequently observed, especially on the legs. Eruptions like those occurring in peliosis rheumatica and in erythema multiforme are also encountered. These phenomena are usually an indication of grave disturbance of the general nutrition, and materially affect the prognosis.

Changes in the blood itself are found only in amyloid degeneration, in extensive tubercular pleurisy, and in protracted continuous fever. As a rule, there is more or less pronounced poikilocytosis or slight leucocytosis; the percentage of hemoglobin varies, and corresponds in general to the external signs of anemia.

A rare complication is *paralysis of the vocal cords.* As in aneurysm of the aorta, it develops on the left side, and is not by any means always due to the pressure of the exudate, as it may be the result of inflammation of the perineural connective tissue and compression of the nerves by bands of connective tissue or swollen lymph-glands.

It is somewhat remarkable that swelling of the bronchial lymph-glands very rarely occurs in chronic uncomplicated pleurisy, especially when tuberculosis is excluded.

In addition to the complication of chronic tuberculosis which, as has been said, occurs both as the cause and result of pleuritic disease, we also occasionally meet with *acute miliary tuberculosis* in the course of a pleuritis. It appears to be particularly apt to develop when a pleuritic effusion undergoes rapid absorption. It is, of course, possible that instead of resorption of the exudate being the cause of miliary tuberculosis, the sudden absorption of the effusion is merely a sign that the general disease has developed in the organism and is making its influence felt on the metallic processes. If we accept the modern doctrine of tuberculosis, based exclusively on the discoveries and views of Koch, it will be difficult to admit that resorption of an exudate that does not contain the specific bacilli can produce miliary tuberculosis. We must, therefore, assume that the bacilli were already present in a diseased focus of the compressed lung, and that the invasion of the general circulation by the organisms and their propagation are facilitated by the sudden removal of pressure from the lung. If this is true, puncture in such cases would be absolutely contraindicated.

If, however, we adopt a view which is possible without doing violence to the facts, that the development of numerous minute nodules in the organism may be due to the action of other irritants besides tubercle bacilli, or any form of micro-organisms within the vascular channels,—that is to say, if it is admitted that the formation of miliary tubercles, like that of other minute inflammatory foci, is not always the expression of a specific cause like the tubercle bacillus,—the production of miliary tuberculosis may be attributed to the sudden entrance into the body of substances chemically different, or of other organisms.

The doctrine that acute miliary tuberculosis is absolutely fatal does not appear to me quite unassailable; for, aside from the theoretic possibility of the organism, which, as we know, can recover from the severest pyemic processes, being able to survive to a certain extent the development of minute miliary tubercular foci,—in other words, repair the histologic structure after the irritant has been overcome,—I have observed a number of recoveries in cases where all the phenomena of the symptom-complex clearly indicated the existence of miliary tuberculosis.

Although such cases are nowadays regarded as conclusive, because they lack the confirmation of postmortem examination, it would be the height of skepticism absolutely to deny the possibility of recovery from disseminated tubercular processes, since recovery from any general infection or any infectious disease represents a regeneration of diffuse diseased foci, and no one doubts the possibility of recovery from local tuberculosis or of the conversion of tubercular foci into connective tissue.

XI. DIAGNOSIS.

The rational employment of exploratory puncture not only affords a means of determining at once the existence or non-existence of an exudate, but also secures direct information as to the nature and amount of material present.

Hence, if exploratory puncture is methodically carried out, an error in diagnosis is possible only when the exudate occupies the base of the lung or when it is encapsulated in the interior of the lung between two lobes. Even in the latter case it is often possible to obtain a positive result provided the exudate touches the surface at a single point, if the proper kind of needles are employed, and the right spot is selected after a careful consideration of all the diagnostic factors. It is particularly the results of *palpatory exploratory puncture* that, in connection with the physical signs and the temperature curve, in the great majority of cases yield positive information in regard to the presence of plaques or even of tumors.

Even this method, however, may sometimes prove inadequate to solve the mystery of an obscure case, since, as has already been mentioned, **diaphragmatic pleurisy, interlobular pleurisy,** and the complication of **pleurisy with an accumulation of pus below the diaphragm** present the greatest diagnostic difficulties. It must not be forgotten that a single symptom, even though it be a very important one, does not suffice to explain the entire condition of affairs; nor can prognosis and treatment be based on a single symptom. It would be wrong to present the results of experience in a few rare cases of great diagnostic difficulty as dogmatic principles, as the clinical material at the command of even a single observer constantly grows, and fresh combinations are continually encountered that tax the powers of the keenest diagnostician. And it would be equally wrong to generalize from the results so obtained, since it is a common experience that deductions made in one case leave one completely in the lurch in the next one, although it may appear to be in every respect similar. I shall, therefore, not attempt to enumerate all the conditions that may be confounded with pleurisy, confining myself to the more important ones, the more so since, owing to the introduction of exploratory puncture, many of the earlier methods of diagnosis have fallen into disuse.

Thus it is difficult, not to say impossible, to distinguish a certain form of *extensive pneumonic infiltration* from a large pleuritic exudate solely by the symptoms elicited by physical methods, whereas the distinction immediately becomes possible if exploratory puncture is resorted to. The diagnosis by means of physical examination is obviously difficult if the infiltration, instead of yielding the usual moderate dulness with an added tympanitic element, produces an absolute flatness; if the breathing, instead of being bronchial, is diminished in intensity or quite inaudible, as in large fluid exudates; if râles are absent, and the characteristic sputum of pneumonia as well as bronchophony and increased fremitus, are not present. Other symptoms, such as insidious onset and slow progress of the disease, absence of the initial chill, and distinct enlargement of the lower portions of the thorax, afford a very uncertain basis for the diagnosis of pleurisy. Now, however, all these difficulties are removed at once by exploratory puncture.

And, conversely, the diagnosis may be difficult if the pleuritic exudate, as is the case in children and young individuals, or when the exudate is thin and purulent, is accompanied by loud bronchial phenomena and considerable increase in the fremitus; in such a case also exploratory puncture affords the only means of arriving at a positive diagnosis.

Again, the distinction between a fluid and a plastic exudate, which is so important for the prognosis and treatment, can only be arrived at by the aid of exploratory puncture. All other signs proposed as characteristic of encapsulation and membrane formation are absolutely illusory.

It is doubtful whether purulent sputum derived from the pleura can always be readily distinguished from the contents of bronchiectatic cavities or the product of a profuse purulent bronchitis. The higher the specific gravity and fat percentage of the sputum, the greater the probability in favor of the former assumption; but it must be remembered that pleuritic pus may be very thin, and bronchial pus, on the other hand, become quite tenacious by inspissation. The figures given by various authors for the specific gravity of pleural fluids vary within very wide limits; in tuberculosis with abundant expectoration the sputum may range between 1010 and 1025, and even higher figures have been reported. At all events, a simpler plan than calculating the percentage of fat or albumin contained in the sputum to determine whether the pus is derived from the pleural cavity, consists in making an exploratory puncture of the pleura and thus obtaining the means of directly comparing the pleuritic pus with the sputum.

According to the generally accepted doctrine, **hydrothorax** can be confounded only with a double exudate, as it is said that transudation is always bilateral. The matter is not quite so simple, however, for an exudate may be superadded to a transudate, or vice versa, and unilateral hydrothorax may occur in many forms of liver disease, and even in heart disease, if the venous flow from one pleural cavity—usually the right in liver disease—encounters unusual resistance.

Chronic myocarditis, which in exceptional cases does not manifest itself by marked general symptoms, is very often accompanied by a right-sided, slowly increasing pleuritic effusion, to which after a variable length of time edema of the legs, enlargement of the liver, and only much later a left-sided pleuritic transudate may be superadded. These effusions, as indicated by the course of the disease, are not to be classed exclusively in the category of pleurisy nor in that of hydrothorax; for, while the specific gravity is exceedingly low, the fluid is quite rich in morphologic elements and fibrin shreds, and the effusion is not rarely accompanied by slight evening elevations in temperature.

In most cases the bilateral character of the effusion, the acute development of dyspnea, the high fever, the absence of cardiac and pulmonary disease and edema, and, finally, the result of exploratory puncture will enable one easily to distinguish between hydrothorax and pleurisy.

One of the surest means of distinguishing between hydrothorax and an exudate is based on the investigations of Rosenbach and Pohl,[9] who found that even small amounts of *iodin* or its salts, when administered by the mouth, are recovered in large quantities and within a short time in the transudate, whereas in exudates only a trace is found; by the ordinary methods of analysis—that is, without incineration—iodin cannot be demonstrated in an exudate. The small amount of fluid obtained by exploratory puncture suffices for the test for iodin, and the rapidity with which the drug is absorbed offers a valuable means of determining the nature of the fluid. If the test (consisting, as is well known, of the addition of fuming nitric acid and agitation of the fluid with chloroform) is negative,—that is, if the chloroform at the bottom of the tube is not colored red,—we have to deal with an exudate; but if the test is positive, even after the administration of small doses, the presence of a transudate is proved. If only a trace is found, it indicates a complication of exudate and transudate, which is not so very rare.

It is often very difficult to recognize the **complication of a pleuritic with a pericardial exudate,** especially if the two exudates are not separated by a zone of resonance. As a rule, however, the addition of a pericardial exudate brings about a sudden aggravation of the symptoms, so that suspicion of cardiac disturbance becomes almost irresistible. Dyspnea becomes more severe and goes on to orthopnea, the pulse becomes small, the heart sounds exceedingly weak, and the cardiac action, even in the absence of demonstrable dislocation or compression of the heart, so rapid and insufficient that the veins of the neck suddenly become dilated or show continuous undulation. If, in addition, there is a pericardial friction sound, the diagnosis of complication with pericarditis is practically established.

Although it is in most cases easy to distinguish the friction sound of pleurisy from all other similar murmurs, such as muscular tones and dry râles, there are cases in which, even with the aid of various manipulations calculated to influence the production of the sound, such as coughing and taking deep breaths, it is difficult to render the sound so characteristic as to preclude the possibility of confounding it with râles which, on account of their tendency to change, ought to be readily recognized. In such cases repeated examinations will be necessary.

When auscultation is rendered difficult by the presence of great muscular pain, admitting only of slight respiratory movements, the distinction between ordinary muscular rheumatism and pleurisy may require the application of a moderately strong induced current to the painful areas, the electrodes being carefully moistened. If the pain is due to muscular rheumatism, it is, as a rule, much diminished after one or two strong contractions, while pleuritic pain is not affected or, if it is, becomes aggravated. Due care must, of course, be exercised in employing this method, as well as massage of the painful region, which is similar in its effect, so as to spare the patient unnecessary pain.

Abscesses in the thoracic wall and **peripleuritic abscesses** (see page 895) cannot always be distinguished from empyema necessitatis. The diagnosis becomes easy if the abscess is distinctly circumscribed and forms a permanent tumor the outlines of which coincide with the limits of dulness. If, however, the dulness extends beyond the prominence, the distinction between encapsulated pleurisy and abscess of the thoracic wall is difficult, not to say impossible. [See "Sub-pectoral Abscess," Musser, "Trans. Ass. Amer. Phys."]

The diagnosis becomes exceedingly difficult if the abscess is complicated with an effusion, or if, on account of edema of the thoracic wall, the percussion note is much impaired. But even in such cases the striking redness of the part, and the tenseness and sensitiveness of the tumor usually decide in favor of an abscess, as there is rarely much redness or swelling in empyema necessitatis. At most there is a bluish or bluish-red discoloration of the center of the prominence, and the surface temperature over the entire fluctuating region, in contradistinction to what is observed in acute abscess, is not materially elevated.

In addition, the upper limit of dulness in exudate is, as a rule, much more regular in outline than in infiltrations of the thoracic wall.

The fact that abscesses of the thoracic wall, not in communication with the pleural cavity, do not affect the respiration, or at most become enlarged and somewhat more tense during inspiration, is of comparatively little diagnostic value; for the occurrnece of regular periodic expansion and contraction, synchronous with respiration, is comparatively rare even when there is an accumulation of fluid within the pleural cavity.

A more important sign is one pointed out by Wintrich,[1] which is the presence of a circular indurated border surrounding a depression felt underneath an abscess in the thoracic wall, similar to the indurated border felt in inflammation and abscess on the skull. This depression, surrounded by the characteristic indurated border, does not, of course, indicate a perforation in the bone any more than in the case of the skull.

The fact that a tumor diminishes under uniform pressure with the hand, which is regarded as a positive sign of abscess, does not necessarily indicate the existence of a communication with the pleura, for the contents of a pleural hernia—that is, a uniform bulging of the pleura without perforation—can be pushed about in various directions by the application of considerable force.

This hernial variety of empyema necessitatis is closely related to *empyema pulsans* (see page 896).

Abscesses of the thoracic wall may exhibit comparatively marked pulsation, and a pulsating empyema is, I believe, nothing more than an abscess at the level of, if not within, the thoracic wall, which is formed when the intercostal spaces become dilated and the pleura bulges outward like a hernia without undergoing necrosis.

According to the generally accepted view, empyema necessitatis is produced only by *necrosis of the costal pleura.* This view, however, does not hold in every case, as will be shown by a case quoted later on. It is very probable that a bulging of part of the thoracic wall is much more frequently due to a kind of hernial protrusion of the yielding costal pleura, made possible by yielding intercostal spaces and great width of the ribs.

As a necessary consequence of arrosion and tension, the pleura eventually undergoes necrosis in these situations even before rupture on the external skin takes place. Nevertheless a mere spheric prominence of the chest-wall containing pus must not be regarded *à priori* as a positive sign of necrosis of the pulmonary pleura.

It is important to point out that in pleurisies of suspected **tubercular origin** the apex of the diseased side is very frequently found to be quite healthy, while the opposite apex shows distinct signs of a still existing or former tuberculosis, such as slight dulness, bronchial breathing, and râles. As it is proved by many postmortem examinations that the exudate in tubercular or phthisical processes is much more frequently found on the sound side, where the production of caseation foci or tubercles is least active, a careful examination of the apex on the apparently healthy side is earnestly recommended in all cases of pleural effusion.

The cause of this peculiar behavior of pulmonary tuberculosis and pleural disease is not easy to find. Possibly the already diseased lung, having for some time been subject to irritation, is less capable of producing a fluid exudate, or else the pleura over the relatively or absolutely healthy lung is more susceptible to certain irritants.

According to our observation, a small pleuritic effusion, or an extensive friction sound resembling crepitant râles associated with rusty sputum and not accompanied by other local changes, are among the most important symptoms of **miliary tuberculosis,** especially when occurring in very dyspneic and cyanotic individuals. Hence, whenever the dyspnea and cyanosis are out of all proportion to the amount of the exudate and the physical signs generally, and the patient shows no signs of cachexia, miliary tuberculosis should always be thought of and the eye-grounds subjected to a careful examination.

To recapitulate, the following circumstances are in favor of the presence of tuberculosis: Plastic pleurisy; hereditary predisposition; tubercular disease of the lung tissue; insidious onset with grave anemia; irregular, moderate, and distinctly remittent fever lasting for some time; very intense dulness, especially over the apices, with diminished vesicular or indefinite respiratory murmur; regular night-sweats; and, finally, a practically negative result from exploratory puncture when, in spite of intense and diffuse dulness, only a few drops of a perfectly clear or hemorrhagic fluid are obtained. The diagnosis is further confirmed by a swelling of the supraclavicular or cervical lymph-glands. The diagnosis of malignant tumor in the pleura is based partly on the above-mentioned symptoms of cachexia, contraction of the posterior and lateral portions of the thorax, the character of the exudate which is distinguished by an abundance of dark brown, liquid blood, fat, degenerated cells or free fat droplets visible to the naked eye (Quincke), and partly on the sudden enlargement of the supraclavicular or axillary lymph-glands.

The importance of determining the presence of particles of tumors in the fluid by macroscopic and microscopic means hardly needs to be mentioned. But this demonstration is not without its difficulties, and I agree with A. Fraenkel,[3] who affirms that only those particles which are actually due to the degeneration of a neoplasm—usually a carcinoma, since sarcoma does not ulcerate—should be utilized in the diagnosis.

Unfortunately, tumor cells are not so characteristic as to preclude confusion with nests and villi of epithelioid cells which occur in chronic inflammations, the large vesicular nuclei of such cells closely simulating the nuclei of carcinoma cells.

Not all accumulations of large polymorphous cells, such as described by Ehrlich[4] as epithelial fatty elements containing vacuoles and surrounded by a villous border, are to be regarded as derived from tumors; and while it is true that such products of fatty metamorphosis, which form the chief basis for the chylous transformation of the exudate, are exceedingly common in carcinoma and tuberculosis of the pleura, they are neither a positive sign of malignant disease nor a pathognomonic sign of tuberculosis.

For this reason I do not attach much diagnostic value to fat granules and fat cells (Quincke[6]), nor to a chylous condition of the exudate. Fatty change is merely a sign of an unusually rapid degeneration of the inflammatory products; in other words, of intense irritation of the tissues and rapid nutritive disturbances.

The **diagnosis of tumor** cannot, therefore, be made by the results of an exploratory puncture, as true particles of tumor are unable to pass the narrow needle of a Pravaz syringe. When large masses of fluid are evacuated through the wide cannula of a trocar, it is of course possible to obtain larger particles which may settle the diagnosis.

The presence of a tumor is indicated particularly by the increased resistance felt on thrusting the needle through the pleura, especially in the absence of fever or cachexia which practically excludes the formation of simple inflammatory plastic exudate.

Unverricht[2] observed the development of malignant tissue in the puncture wounds in many cases of endothelioma of the pleura. The dark red color of the evacuated fluid, which, according to A. Fraenkel,[3] presents the appearance of the blood as seen in blood-letting, may determine the diagnosis.

Purjesz[9] has expressed himself to the effect that the intercostal spaces in tumor of the pleura are not widened, but retracted on the diseased side; but this rule does not apply to all cases, as the retraction may also be very marked in the presence of a very thick plastic exudate, the product of chronic inflammation without malignant growth.

A detailed description of **echinococcus of the pleura** has been contributed by Maydel.[3] The diagnosis can practically always be made with absolute certainty by exploratory puncture, hence all other diagnostic signs are of little importance. The very possibility of the existence of an echinococcus tumor, which can only be permanently and safely removed by operation, is an earnest plea for the importance of exploratory puncture, which must, therefore, be employed in any case that presents the least element of doubt.

Neither an irregularity in the area of dulness, nor the ill-defined changes in the respiratory murmur, nor a partial bulging of the chest-wall, nor the temperature, nor the condition of the glands, affords any positive data for the differential diagnosis between malignant, incurable tumors and the relatively benign, because operable, echinococcus. If, for fear of suppuration taking place in the sac after puncture, a diagnosis were delayed until perforation into the lung revealed the presence of echinococcus vesicles in the sputum, the time when operation offers the best hope of success would be missed. A negative exploratory puncture does no harm to the patient, whatever the condition of the pleura, while a positive result always affords some information of value both for prognosis and for treatment. The danger of suppuration of an echinococcus sac taking place after puncture need not be considered, since the finding of vesicles or hooks, or a certain peculiar nature of the fluid which is characteristic of these cysts, demands immediate radical operation.

It is also important to determine the seat of the tumor, since it may develop outside of the pleural cavity, and may only simulate a primary affection of the pleural cavity on account of complication with disease of the pleura or on account of displacement of organs. The case is similar to that of subphrenic and paraphrenic abscesses, and the same diagnostic signs apply. At any rate, repeated exploratory puncture will afford a certain amount of information, if only by revealing the depth to which it is necessary to penetrate to reach the pus cavity.

[Cary and Lyon * report a case of **primary echinococcus cyst of the pleura.** The salient features of the case are: Clinical diagnosis of chronic encysted pleural effusion, based on the physical signs and the aspiration of an albuminous fluid loaded with cholesterin crystals. Operation. Lung free; no adhesions, no effusion; removal from the pleural cavity of a mass of collapsed cysts resembling echinococcus cysts. The cyst walls were thin, hyaline, without the characteristic lamellation, and infiltrated with cholesterin crystals. The diagnosis was finally established by the finding of two hooklets. The patient, a mulatto, thirty-one years of age, had never been outside the United States. The termination was fatal. The number of cases of primary echinococcus cysts of the pleura so far reported is 41. The prognosis is practically fatal unless radical operation is resorted to. The 31 cases of primary and secondary echinococcus cysts of the pleura collected by Neisser all had a fatal termination. Death is due in part to purulent changes in the cysts and in part to toxemia induced by absorption of the fluid after rupture of the cysts.

Cytodiagnosis.—Wolff regards a progressive increase in the number of lymphocytes, determined by periodic tapping and examination of the fluid, as indicative of a tubercular etiology. Tuberculous exudates at first contain more polynuclear cells, later the lymphocytes become more prominent. Acute tuberculous exudates contain polynuclear and epithelial cells. Widal and other French writers also mention the abnormal increase of lymphocytes in tuberculous cases, and the rare presence of endothelial cells in pleural fluids of tubercular origin.*

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* *Amer. Jour. Med. Sci.*, October, 1900.

Patella, on the other hand, denies that these so-called lymphocytes have anything in common with the blood, and sees in them the altered nuclei of endothelial cells. They are found in any pleural or peritoneal effusion that has existed for some time, just as, conversely, endothelial cells are found in all recent effusions, including tuberculous exudates. Cytodiagnosis, if Patella is right, is chiefly if not solely of use in determining the age of an effusion, and gives no information in regard to the nature and etiology of the morbid process. †

Warthin ‡ was able to diagnose sarcoma of the pleura by means of spindle cells found in the fluid obtained by aspiration. The diagnosis was confirmed by autopsy. Warthin studied the morphologic elements found in pleural effusions in various conditions. In the early stage of acute serous pleurisy he found large numbers of endothelial cells in masses and isolated, or in groups of two or three. The cells were vacuolated. In the later stages spindle forms are abundant. In acute fibrinous pleurisy, in addition to vacuolated endothelial cells, he found numerous irregular spindle-shaped cells, probably fibroblasts. The sediment in tuberculous cases differed in no respect from that found in acute fibrinous pleurisy. Warthin concludes that the presence of numerous cell-division forms in the cells of the sediment is strong evidence of a neoplasm, as mitoses are rarely found in the cells of purely inflammatory exudates.

Klein states that the appearance of eosinophile cells in the exudate is followed by their appearance in the blood, where, however, they increase at a much more rapid rate. They are found in large numbers in hemorrhagic sputum and in the exudate of tuberculous pleurisy and peritonitis. He believes that the presence of eosinophiles in diseased tissues is the result of active hemorrhage or extravasation of blood. The cells are originally neutrophile leucocytes which have taken hemoglobin or red blood-cells into their bodies and have thus converted their neutrophile granules into eosinophiles. They represent a function, not a pathologic condition. Petrowsky and Zaleski severely criticize Klein's conclusions, and point out that, according to his reasoning, eosinophile cells should occur in all cases of internal hemorrhage, contusions, fractures, and infantile lobar pneumonia, and should not be present in such large numbers in asthma.§—Ed.]

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* *Berl. klin. Wochenschr.*, 1901, Nos. 5, 34, and 45; 1902, No. 6, abstracted in *Edinburgh Med. Jour.*, April, 1902.

† *Deutsche med. Wochenschr.*, vol. xxviii, No. 16, 1902.

‡ *Medical News*, October 16, 1897.

§ "American Year-Book of Medicine," 1900, page 114.

XII. SPECIAL FORMS OF PLEURISY.

1. PLEURISY IN CHILDREN AND METAPNEUMONIC EMPYEMA.

The diagnosis of pleurisy in children, which, on account of the alleged peculiar course of the disease, has been extensively discussed by various authors, does not, in our opinion, present any peculiar difficulties if the examiner exercises ordinary skill and attention, and resorts to the use of exploratory puncture.

That the **symptoms** are usually less uniform—either more violent or more latent—in children than in adults, that the pulse, temperature, and respiratory rate exhibit great variations, will not surprise the experienced physician who has had abundant opportunity to observe how differently children react in all diseases.

Some authors attach a good deal of importance to a *peculiar type of breathing* in the pleurisy of children, which, for my part, I have never been able to observe. Indeed, the lack of specific signs is even greater than in adults, and the most painstaking general and physical examination, complemented by exploratory puncture, is necessary to determine positively whether any exudate is present. The dulness and dislocation of organs is often less pronounced in children than in adults, chiefly because very large exudates are, as a rule, only met with in older children or in very badly neglected cases. Nevertheless I have seen quite extensive serous, and especially purulent, exudates in small children.

As a rule, the symptoms are such as to make it impossible to distinguish, by *physical signs* alone, small exudates from the ordinary bronchopneumonic consolidations. Even in cases of empyema in children loud bronchial breathing is the rule, and diminished breath sounds the exception. The only condition which positively excludes any degree of pleurisy at once is the presence of numerous râles. *Friction sounds* are exceedingly rare in small children, and it may here be pointed out that very light percussion must, as a rule, be used in cases of small exudates in children to determine the limits of dulness with accuracy or, in fact, to obtain any difference in sound at all.

Quite recently attention has been called to the subject of **metapneumonic empyema,** especially in a work of Netter.[3] The condition occurs most frequently in children and young persons, say, up to the fortieth year, and usually, though not always, follows a pneumonia that has not ended by crisis.

The cases often occur in small epidemics. After a short afebrile period the pleuritic process is ushered in by a considerable rise in temperature. The exudate under these circumstances is quite thick and contains much fibrin and numerous pus corpuscles. Although the exudate is frequently encapsulated, or at least surrounded by dense adhesions, *perforation* into the lung is not a rare event. Whether there is a special disposition on the part of the lung to purulent disintegration, or whether empyemas of this kind are in fact the expression of a superficial form of pulmonary abscess, it is impossible to determine. Elastic fibers are only rarely found in the sputum, which in most cases is exceedingly scant.

The *fever* is usually continuous, chills are rare except during the initial rise in temperature, and the process very rarely goes on to edema of the chest-wall.

The *mortality* in this form of empyema is very low. In operative cases the mortality, according to Netter, is to the mortality in empyema due to other causes as 2.3 is to 25. I do not believe that this proportion is correct, however, since even in the ordinary uncomplicated form of empyema the mortality barely reaches 5%.

Indeed, it seems to me that too much importance has been attached to metapneumonic empyema as a nosologic entity, since acute inflammation of the lung is quite frequently accompanied by a cellular pleuritic effusion containing an abundance of fibrin which, however, hardly deserves the name of empyema. I have often obtained a few drops of this purulent fluid with the exploratory needle, and it usually turned out that I had to deal with an ordinary and insignificant complication of pneumonia with fibrinous pleurisy.

The cause of the suppurative process in all uncomplicated cases is said to be A. Fraenkel's pneumococcus. It is to be remembered, however, that uncomplicated empyema is rare, and the exudate, therefore, is often found to contain other pathogenic micro-organisms. The relatively benign character of metapneumonic empyema and the empyema of children has by some authors been attributed to this difference in the bacteriologic etiology, on the ground that the pneumococcus is possessed of exceedingly low powers of resistance.

Lemoine[2] distinguishes between peripneumonic (concomitant) and metapneumonic (secondary) pleurisy,—the latter preferably attacking debilitated patients,—and assumes that the former variety, even when the exudate is large, rapidly undergoes involution after the pneumonia is cured, while in the latter variety the duration of the disease is usually protracted.

[**Pleuritis in the Newborn Infant.**—According to Steele,* idiopathic pleurisy is exceedingly rare in infants. In addition to one case of his own, he collected 24 from the literature and refers to 14 collected by Hervieux, making a total of 39 cases of idiopathic pleurisy in newborn infants. The condition is without exception merely part of a general infection with common pyogenic micro-organisms. The morbid anatomy is that of septic pleurisy; the exudate may be fibrinous or sero-fibrinous, purulent or hemorrhagic. Staphylococcus aureus and albus, and Streptococcus are the commonest causes. The symptoms consist of fever (103° to 104° F.—39.4° to 40° C.) with intermissions, although the temperature may also be normal; rapid loss of weight; refusal to take food; often icterus, and sometimes purpura.

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* *Philadelphia Med. Jour.*, September 17, 1898.

Hemorrhages occur from the umbilicus, from the intestines, and from the skin. The cry is said to be whimpering or whining and quite characteristic. There is commonly diarrhea; vomiting may or may not be a symptom. Death takes place in collapse with subnormal temperature. The physical signs are vague and often very misleading. The prognosis is extremely bad; a temperature above 102.4° F. (39° C.) is always a fatal sign. The disease occurs in children of septic mothers. Infection takes place chiefly through the umbilical wound, where suppuration is difficult to detect. Infection by the mother's milk through the gastro-intestinal tract is at least possible, as evidenced by the fact that puerperal infection of the newborn is frequently associated with gastro-enteritis. Steele insists that a mother who shows signs even of a mild sepsis should not be allowed to nurse her child.—Ed.]

2. INTERLOBAR PLEURISY.

Interlobar pleurisy, or, in other words, the accumulation of a serous or purulent exudate between the apposed surfaces of two lobes of the lung, does not differ, in my opinion, from the other forms of encapsulated exudates, except that, as the dulness does not spread out on the chest-wall, the *diagnosis* in interlobar pleurisy presents greater difficulties.

The difficulties incident to *operative treatment* are even greater. In serous exudates the site of puncture and the depth to which the needle must penetrate, in purulent exudates the proper point for the incision and the finding of the pus cavity, are difficult problems. It is true that a serous interlobar exudate rarely attains such proportions as to make puncture an urgent necessity. We may, however, have complications of small interlobar collections of pus with larger, freely movable serous exudates which obscure both the diagnosis and the effect of treatment, as the fever curve is influenced by both forms of inflammation.

Laennec himself demonstrated the *morbid anatomy* of pulmonary abscess and interlobar pleurisy. Various French authors Perrier, Martinez-Mesa, and Pailhaus, and quite recently Potain, who discusses the differential diagnosis between interlobar pleurisy and large cavities in the lungs—have devoted a good deal of attention to the question.

The *etiology* in cases of this kind is not always certain; sometimes they follow an acute pneumonia, again they may be the result of small tubercular foci, or they represent a renewal of an old pleuritic process, as indicated by the fact pointed out by D. Gerhardt, that old adhesions of the pleura were found at the autopsy in all the cases observed. In view of the close connection between this form of pleurisy and pulmonary abscess, rupture into the lung with a subsequent development of a pneumothorax is at times to be expected. As a matter of fact, however, such an event rarely occurs, and a great number of cases recover without any complication.

The *symptoms* of encapsulated pleurisy resemble those of pulmonary abscess so closely that I do not believe it is ever possible to decide *in vivo* whether there exists a pulmonary abscess accompanied by pleurisy or a primary encapsulated purulent pleurisy. Such a form of pleurisy should always be thought of if the strength rapidly begins to fail, hectic fever and chills make their appearance, there is a circumscribed zone of dulness surrounded by resonant lung tissue, and the patient suddenly begins to expectorate abundant purulent or even putrid material, an event which often marks the beginning of recovery or repeats itself periodically until the patient is completely exhausted. But it is readily seen that the above-mentioned symptoms are ambiguous, and do not in the least aid us in distinguishing primary pleural disease from disease of the lung tissue.

The fact that **interlobar empyema** usually runs a favorable course has led D. Gerhardt [10] to advise delaying operative intervention even after the occurrence of perforation. According to my experience, however, it is best to perform exploratory puncture as soon as possible after the appearance of such pleuritic symptoms as make it possible to determine approximately the seat or direction of the purulent focus, and to make an incision as soon as the presence of pus has been positively determined.

I shall relate an instructive case of interlobar empyema in which these principles were observed both in the diagnosis and in the treatment. The therapeutic principles in complications of pulmonary abscess with empyema or pleurisy will be given below in connection with a very complicated case (see page 946).

CASE III.—*Empyema during the Puerperium. Encapsulation of the Pus in the Fissure between the Upper and Lower Lobes. After thoracocentesis it became necessary to open the cavity by means of a puncture.*

Mrs. X. after her first confinement was seized with chills, and shortly afterward presented the symptoms of a left-sided pleurisy, the dulness extending from the posterior surface to the middle of the scapula, and from that point by an abrupt descent to the lateral wall of the chest. As the fever, chills, and dyspnea continued and the patient rapidly lost strength, I was called in consultation, and as all the signs appeared to indicate a purulent exudate, I performed an exploratory puncture in the lower portion of the left posterior wall of the thorax without finding any pus. A second puncture below the angle of the scapula with a longer needle yielded thin, odorless pus, about 100 c.c. of which were at once evacuated with the aspirator.

As the patient failed to improve in spite of this interference, radical operation was proposed and accepted. The patient was chloroformed and another exploratory puncture performed at the same place, which also yielded pus. Owing to the peculiar position of the exudate, which evidently occupied a circumscribed spot on the posterior surface of the lung between the upper and lower lobes, it was necessary to make the incision in a very unfavorable situation on the posterior surface of the thorax between the scapula and the vertebral column. After another puncture and the evacuation of a small syringeful of pus the incision was made in the seventh intercostal space, but no pus was found when the pleura was opened. Even after a finger was introduced no pus was found, although numerous thin adhesions and plastic deposits were separated.

But as the numerous exploratory punctures positively indicated the presence of a pus cavity at a depth of 6 to 8 centimeters, it was thought that more radical measures for the evacuation of the encapsulated exudate might safely be resorted to, and, accordingly, a trocar was inserted into the opening under the guidance of the finger, whereupon about 400 c.c. of yellow, odorless pus flowed out in a rapid stream.

After the operation the fever disappeared completely and a comparatively rapid recovery ensued, although, owing to the unfavorable position of the wound, the healing was much complicated by slipping of the drainage-tubes and displacement of the scapula. Ten weeks later the patient had entirely recovered except for a small oblique fistula which closed up after a few weeks. There was no deformity of the thorax.

3. PULSATING EMPYEMA.

Pulsating empyema, consisting in a periodic pulsation of the pleural contents, and manifesting itself in alternate bulging and flattening of an entire half, or of a circumscribed portion of the thorax, is a comparatively rare occurrence. As the name indicates, the condition only occurs in purulent exudates, and mostly occupies the region where the anterior surface merges into the lateral wall, or the lateral wall itself. The condition is often associated with empyema necessitatis.

According to the latest statistics, collected by J. G. Wilson, a faint and always systolic pulsation has been observed in a few cases on the posterior wall of the thorax, and three times in the lumbar region.

Rummo makes a distinction between *pulsus esopleuricus*, the visible and palpable positive pulse of a pulsating pleurisy which can be directly recorded by means of a graphic apparatus applied to the thorax, and *pulsus endopleuricus*, which can only be recorded by connecting the pleural cavity with a manometer. The latter form of pulsation is said to accompany any large, left-sided pleural effusion.

For my part, I do not believe that this distinction can be made either theoretically or practically, as *pulsus endopleuricus* is simply an initial stage of *pulsus esopleuricus*, and both forms of pulsation may be produced when the pleura is intact.

The area of pulsation, as a rule, does not exceed the size of the fist, although the pulsatory vibration may extend beyond that limit. The pulsation is almost always systolic, that is to say, the greatest degree of bulging is synchronous with cardiac systole and arterial diastole. During inspiration there is a diminution in the prominence, accompanied usually by diminution of the pulsatory pressure; during expiration the prominence swells, although these conditions are sometimes reversed. Pulsations of this kind are not always, as affirmed by certain authors, a positive sign of necrosis of the costal pleura and undermining of the thoracic muscles, for I have observed them to occur also when the intercostal spaces were unusually wide and the ribs and intercostal muscles abnormally yielding in a certain area, permitting a greater separation of the ribs and increased distention of the soft parts. As soon as this relaxation reaches a certain degree the movement of the heart or the effect of its contractions, which, under normal circumstances, is uniformly distributed over the entire thoracic contents, necessarily makes itself felt in the direction of least resistance, and the corresponding area becomes the site of positive centrifugal (systolic) or negative centripetal (diastolic) waves; that is to say, a prominence or a depression. The systolic distention of the thorax and the impulse of the accompanying increase in the movement of the blood directed downward and to the left produces a centrifugal wave, while the diastolic increase in the venous flow produces a centripetal wave.

The prominence can be diminished by pressure with the hand whenever there is marked edema of the thoracic wall or a communication exists between the thoracic wall and the pleural cavity on account of necrosis of the pleura. As a rule, external pressure merely diminishes the pulsation, and thus simulates a diminution of the prominence. The movements of the thoracic wall are increased in all forms of pulsating empyema.

Osler[10] has pointed out that several pulsating tumors may be present in pulsating empyema, and that rupture may in rare cases take place in the back, while in intrapleural pulsation rupture occurs only in the anterior or lateral walls.

From the fact that the pulsation is removed by the evacuation of even a small quantity of fluid Bouveret[1] concludes that pulsation is dependent on a certain degree of tension, a view which agrees with what I have stated above.

Comby's theory that the pulsation occurs only in compression of the lung and when adhesions with the pericardium are present,—that is to say, under conditions which allow the movements of the heart to be directly transmitted to the pleural fluid and to the thoracic wall,—does not appear to be tenable, as the retraction of the lung is of minor importance compared to distention of the thoracic wall and increase of cardiac activity.

Osler[10] states in regard to the history that although older authors (Baillon, LeRoy, Pelletan) described tumors pulsating synchronously with the heart, the first communication in reference to diagnosis of pulsating empyema came from L. MacDonnell, an assistant of Graves and Stokes.

That a pulsating empyema should be mistaken for an *aneurysm* does not seem possible with our present knowledge of diagnosis. In ambiguous cases any doubt can, as rightly remarked by Osler, be at once removed by performing an exploratory puncture with a very small needle.

[**Chylothorax.**—Hahn* describes a case of chylothorax which resulted from fracture of the tenth dorsal vertebra, caused by the passage of a heavy wagon over the patient's body. A right pleural effusion, at first hemorrhagic but soon becoming almost entirely chylous, developed shortly after the injury. During a period of fifteen days 30 liters (quarts) of chylous fluid were taken from the pleura, and at the autopsy 7 liters more were found. Chylothorax may be said to present two degrees of severity, according as the main trunk or only branches of the thoracic duct are involved in the injury. Cases belonging to the first group are necessarily fatal; in the second group there is likelihood of recovery. The severity of the injury is recognized by the rapidity with which the exudate is formed.—Ed.]

4. PERIPLEURITIS.

That any attempt to classify clinical symptom-complexes from a mere anatomic point of view accomplishes no practical purpose and fails to take due account of the logic of facts is perhaps best shown by the definition of peripleuritis.

By peripleuritis (more correctly parapleuritis) is meant a *primary* —in a certain sense spontaneous—inflammation and suppuration of the layer of connective tissue interposed between the costal pleura and the muscular layer of the chest-wall, the so-called subserous (subpleural) connective tissue. The secondary inflammatory affections and suppurations in this region, which, as is well known, not infrequently occur after traumatism, or as the result of disease of the ribs or vertebræ and after pleural affections, must be strictly distinguished from true peripleuritis or inflammation without demonstrable cause.

That these propositions fail to afford sufficient grounds for diagnosis will be admitted by every one, and hence what I have said in the second edition of Eulenburg's "Real-Encyklopädie," in the article on "Peripleuritis," will, I think, meet with general acceptance.

It is evident that on the basis of this definition the **diagnosis** of the affection *intra vitam* must always be very uncertain on account of the impossibility of excluding the presence of small, latent foci in the neighborhood from which the process of infiltration and suppuration might have spread to the peripleural connective tissue, as the latter is naturally poor in blood-vessels and of low vitality. Until the question is settled by an autopsy it is impossible to decide between a peripleuritis in the strict sense of the word and a mere secondary peripleuritic abscess, a term which I prefer to use to distinguish the condition from the so-called idiopathic affection.

This definition of peripleuritis as an idiopathic disease seems to me very forced, based, as it is, on the etiologic factors and the pathogenesis, and the diagnosis is further complicated by the difficulty of determining whether an inflammation within the thoracic wall has begun in the subserous connective tissue and not in the muscles or intramuscular connective tissue.

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* *Deutsche med. Wochenschr.*, June 22, 1899.

In any case, then, the difficulties encountered in the diagnosis of peripleuritis are unquestionably great if we adhere strictly to the above definition and attempt to exclude traumatism or any other inciting factor in the neighboring tissues. As it is not of the least prognostic importance to determine whether we have to deal with a primary or secondary disease,—since operative evacuation of the pus is necessary in any case, and will therefore clear up any doubt that may exist in regard to a possible connection between the suppuration and a primary disease in the immediate neighborhood,—peripleuritis need not be considered as an idiopathic disease, and at most possesses a certain interest for diagnostic and prognostic reasons.

Accordingly, it is not because of any systematic importance of the condition, but only out of regard for the men (Wunderlich, Bartells, Billroth) who have attempted to define peripleuritis, that we give a short exposition of the most important points in connection with this subject. Omitting for the present secondary pleuritis,—that is to say, abscesses developing after disease of neighboring organs or in pyemia,—I may mention that the investigations of the past decades in actinomycosis have resulted in explaining to some extent the etiology of many hitherto obscure cases of primary abscess formation. The chronic forms with numerous communicating fistulæ are practically always dependent on *actinomycosis*.

The abscess differs in nowise from similar purulent affections in other portions of the body. It contains the usual forms of pyogenic cocci and granules, and, if it is dependent on actinomycosis, the characteristic fungi. Its external wall is formed by the soft parts of the thorax, the internal wall by the thickened but not always obliterated pleura. Sometimes a pocket is found along the diaphragm, and occasionally even the thickened peritoneum forms a portion of the abscess wall (Riegel). Fistulæ running in various directions and communications between the various parts occur.

Peripleuritic inflammation appears to occur at *any age*, and particularly in the *male sex*.

The disease is pronounced, and acute cases usually begin with a *chill;* the subsequent course of the fever presents nothing characteristic. Special importance is attached by some authors to the continuous, deep-seated, boring *pain* which persists even after the occurrence of suppuration. Other signs, such as the absence of congestion and of dislocation of neighboring organs, of cough and sputum, are of little diagnostic value, as these phenomena are not always present even in pleurisy.

A more important symptom is great *interference with breathing,* which occurs early as a result of the unusually severe pains in a large area of the thoracic wall, although this sign also occurs in pleurisy. Another sign is a peculiar *circumscribed tenderness* to pressure on the surface of the thorax and a doughy swelling of irregular outline.

As soon as the abscess has attained a certain size and becomes perceptible to the touch, the diagnosis is readily made, sufficient data being afforded, on the one hand, by the evident presence of inflammation and bulging of the outer wall, and, on the other hand, by the negative findings in the lung and pleural cavity.

According to Bartels, the outer wall of the abscess grows tense during inspiration and relaxes during expiration, a condition which is also found in large pleural effusions.

The presence of distinct *fluctuation* is, generally speaking, more characteristic of abscess, although fluctuation may be produced in pleural exudates and hydrothorax if the intercostal spaces are unusually wide. If the abscess is deep and embraces a large portion of the thoracic wall, it can usually be distinguished from an extensive pleurisy by the fact that it does not produce any dislocation of the heart or displacement of the apex-beat; for whenever a purulent accumulation in the pleura is so great as to produce an empyema necessitatis, the signs of a dislocated heart are never absent.

I need not waste any words about the signs obtained by percussion and auscultation in abscess, the movability of the edges of the lung, and the presence of resonant normally functionating lung tissue below and around the borders of the abscess-dulness; these points in the differential diagnosis can, of course, apply only to a free exudate, whereas an encapsulated empyema in process of transition into empyema necessitatis may display the same symptoms as a peripleuritic abscess.

Bartels affirms that pus from the pleura never exceeds 1032 in specific gravity, while the specific gravity of pus from an abscess is much higher. But the specific gravity of an empyema-pus depends on the duration of the disease and the degree of suppuration, and may under certain conditions exceed the limit mentioned.

Again, as in all other cases of pleural disease, *exploratory puncture* and *operation* afford the only means of arriving at an early decision. The depth at which the pus is found gives the first clue to the diagnosis, which is then confirmed by the nature of the pus, as, for instance, when it contains the characteristic actinomyces elements, which, so far as we know, have never been found in cases of uncomplicated empyema.

At all events, exploratory puncture gives definite information in regard to the presence of pus, and at the operation, which is then absolutely indicated, the remaining conditions will be cleared up, since they can be directly investigated by digital exploration of the cavity.

The following complications (or coordinated manifestations) of peripleuritis are the most common: diffuse nephritis, pericarditis, peritonitis, pyemia, necrosis of the ribs, deep abscesses. Perforation toward the exterior is, of course, quite common; perforation toward the pleura apparently occurs rarely, because the layers of the pleura become obliterated by the inflammatory process.

The **prognosis** is unfavorable in protracted cases and when the abscess is a concomitant of pyemia or glanders. The earlier the pus cavity can be opened and the cause of suppuration removed, the more certain is the prospect of cure. The extensive abscesses with accompanying actinomycosis are the most difficult to cure, but even in such cases, thanks to modern surgery, the treatment appears to have been occasionally successful.

The **treatment** is determined by the conditions of the individual case, and should be carried out according to generally accepted surgical principles.

XIII. PROGNOSIS.

The prognosis in pleurisy appears to depend in general less on the intensity of the local, especially the initial phenomena, and more on the patient's condition, his power of reacting, and hereditary disposition.

In the so-called *secondary, non-rheumatic* forms of pleurisy (see page 803) the prognosis, of course, depends chiefly on the basal disease (inflammation in neighboring organs, pyemia, tuberculosis). Nevertheless the local process in the pleura in many cases becomes so prominent that the ultimate outcome is largely determined by it. In persons predisposed to tuberculosis apparently simple forms of rheumatic pleurisy may become chronic; in a distinctly acute pleurisy the process of absorption may suddenly become arrested, or continued remissions and exacerbations of all the symptoms, especially the fever, may occur.

I do not believe that a distinct decision in regard to the progress and duration of the disease can be arrived at within the first two weeks. I have seen a number of effusions beginning with intense dyspnea, fever, and great cardiac weakness run an extraordinarily favorable course; and, on the other hand, have seen cases where the exudate was so slight and the general condition so satisfactory that there was every hope of a speedy resorption eventually turn out very unfavorably. Besides, it often happens that recovery from a first attack, apparently beginning under the most favorable conditions, is interrupted by a much more serious and tedious relapse.

In cases showing high temperatures in the third week, in those which during this time or even earlier present hectic fever or a striking morning remission, in cases in which an inverse type of temperature or irregular fever makes its appearance toward the end or beginning of the third week, and, finally, in cases beginning at once with profuse sweating without distinct lowering of the temperature, the prognosis should be extremely guarded.

Unless there is active absorption in the beginning or toward the end of the third week, the prognosis as regards complete recovery should be distinctly more unfavorable, not only because the exudate, on account of its extent, makes resorption difficult,—since the amount of fluid can be reduced to any desired volume by means of tapping,—but because the delayed appearance of resorption indicates that the inflammatory irritation is abnormally great, the vital energy distinctly impaired, or that there is a permanent weakness on the part of the tissues which cannot be removed by the mere relief incident to mechanical evacuation of the exudate any more than the disturbances in the liver tissue due to cirrhosis can be relieved by evacuating an ascitic fluid. In fact, we have in such cases to deal with the above-mentioned combination of pleurisy with hydrothorax, or, as I prefer to put it, the combination of a maximal chemical irritation of the tissue with a considerable permanent impairment of the mechanical power of transporting lymph and blood.

Whenever the abnormal irritant, be it the exudate or the organic causes of inflammation (bacteria and their products), or even some unknown agent, ceases to exert its influence and the activity of the tissue becomes available for the normal, mechanical part of their work,—that of transportation,—the kind of work performed undergoes a change, and the external work, that of transferring the products of tissue activity to the blood, is again fully resumed.

In *double pleurisy*, in *chylous effusions* due to an extreme degree of fatty degeneration of the contained cellular masses, and in *hemorrhagic forms* the prognosis is always extremely doubtful, because there is in such cases either a great primary impairment of the organism, a fatal constitutional etiologic factor such as carcinoma and tuberculosis, or else the irritation is abnormally great.

An exception is the double serous pleurisy occurring in the course of articular rheumatism and during the puerperium. In this form the prognosis is not always absolutely unfavorable, providing the exudate is not excessive and the patient was in fairly good condition before the attack, and no complications make their appearance. Under favorable conditions I have seen a number of cases of double pleurisy and pericarditis get well, especially when the size of the exudate was greater on one side than on the other. Hence the prognosis, especially in a serous effusion, is not directly dependent on the size of the exudate, but rather on certain local conditions the importance and nature of which are not as yet accurately known.

Spontaneous rupture of an empyema always has an unfavorable effect on the prognosis so far as complete recovery is concerned, partly on account of the possibility of pneumothorax or putrefaction setting in, and partly because, in spite of the evacuation of large masses of pus, the exudate continually tends to reaccumulate on account of obstruction of the sinus. In view of the excellent results obtained by early operation in empyema, an accident like rupture through the bronchi or the chest-wall ought not to take place if all the various methods of diagnosis, particularly exploratory puncture, are employed and properly carried out. The nature of an exudate can now be determined a very few days after the beginning of the disease, and in any case of purulent exudate, unless the amount of fluid is exceedingly small, immediate operation is, in my opinion, indicated.

The percentage of albumin, fat, salts, globulin, and peptone contained in the exudate has been utilized by some authors in the diagnosis and prognosis; according to Bernheim,[6] for instance, a low percentage of albumin in the exudate indicates that the course will be protracted. It does not appear to me, however, that these laborious studies have led to any satisfactory results.

If an exudate containing few cellular elements should rapidly begin to reaccumulate after it has been evacuated, say, at the end of the second week, the prospects of resorption taking place are, as has been said, not very favorable; they are distinctly unfavorable if a second puncture is followed by the same unsatisfactory result.

If an exudate contains abundant cellular elements and is accompanied by high, continued fever, the prognosis in regard to rapid resorption is rather unfavorable. At best, the disease may be expected to last many weeks.

It is to be observed that an exudate containing abundant cellular elements has not by any means the same significance as a purulent exudate, for spontaneous resorption may occur even when the cellular elements are exceedingly numerous, while purulent exudates very rarely heal by absorption. Hence an abundance of cells is not identical with pus formation.

My experience will not allow me to admit that the great advance in bacteriologic examination of the exudate during the past decades has added any material data for the clinical interpretation and management of a case, either as regards the prognosis or the treatment. The mere presence of A. Fraenkel's pneumococcus is not enough to induce me to give a favorable prognosis, or to prophesy spontaneous cure, any more than the presence of several forms of pyogenic organisms necessarily renders the prognosis absolutely bad, since even the most complicated forms of empyema are often rapidly cured by radical operations. [This is not borne out by the experience of American physicians. For them a pneumococcus empyema is of good prognostic import.—Ed.]

The absence of all bacteria from a purulent exudate is not by any means a certain sign of its tuberculous nature, as many authors affirm, nor can tuberculosis be excluded when the tubercle bacillus is absent, as I have already explained.

Sacaze[4] affirms that a positive finding of streptococci always excludes the presence of tubercle bacilli, but even he concludes that the presence of streptococci is by no means a safe criterion of the extent of the purulent process.

One should never be betrayed into giving an unfavorable prognosis in the case of pleurisy on account of alterations in the respiratory murmur over the apex of the diseased lung, such as occur in mild apical affections. After observations extending over many years I have convinced myself that in many cases the upper portion of a lung affected with pleurisy may for a long time present alterations in the respiratory murmur which are due solely to deficient respiratory movements and not to any tuberculous disturbances in the lung itself. A mere diminution of the respiratory murmur, or a slight dulness indicating a diminution in the amount of air contained in the less active lung, is no proof of the existence of apical disease under such circumstances, nor is a faint tympanitic element in the note in the supraclavicular or infraclavicular fossa of the slightest significance as long as any signs of pleurisy remain and the respiratory excursions are deficient. Even the presence of a few isolated râles or a slight roughening of the respiration is of little significance under the circumstances unless the sputum, which in such cases must always be carefully examined, indicates gross disease of the lung.

As the determination of the slightest tubercular changes in the lungs complicating a pleurisy is of the utmost importance, it might be well to point out a few of the sources of error to be avoided in examining the sputum or other pathologic products for tubercle bacilli, especially the errors due to accidental contamination of the preparations. When we first began to examine for tubercle bacilli, I was struck by the fact that I occasionally found a few isolated bacilli in one or two preparations where, according to my opinion, tuberculosis could be positively excluded; and when I once found a nest of bacilli in an undoubted case of simple bronchial catarrh, I came to the conclusion that I had to deal with some accidental contamination. It was not long before I succeeded in discovering two frequent sources of error, one of which is the use of slides and cover-slips that have already been used in preparing specimens of tuberculous sputum; the other, the use of the same staining fluid when the method of staining in glass dishes is employed. In the former case the bacilli adhered to the glass, as I found by a careful inspection of apparently well-cleansed slides; in the other, the tubercle bacilli are contained in the staining fluid used for former preparations of tubercular sputum, and are deposited with the staining fluid on fresh specimens. Since I made this discovery I have adopted the rule: First, to use only absolutely new slides in preparing specimens; and, second, never to stain in glass dishes, but instead to drop the staining fluid directly from the bottle on the microscopic slide after the specimen has been spread out and dried. The specimen should always be rinsed under the spigot or with a water-bottle, never in a staining dish.

Even when all these precautions are observed it should be a rule never to attach too much importance to the finding of isolated bacilli, especially if they are only found in one out of a large number of specimens examined. Of course, when sections of tissue are examined the finding of even a single bacillus, providing it is actually embedded in the tissue, is a very different matter.

The sputum-cups themselves may become the sources of contamination whenever they are not cleansed with scrupulous care. Small particles of sputum frequently adhere to the sides of the vessel, as I have often convinced myself, and may thus become sources of error if the same vessel is subsequently used by a non-tuberculous patient.

That the same sources of error are to be carefully guarded against in the examination of any other secretion such as an exudate needs only to be mentioned.

XIV. TREATMENT.

"Tremendous advances have been made during the past ten years in the treatment of serous pleurisy, as has already been mentioned, especially by the introduction of the sensible practice of removing the exudate by means of thoracocentesis, and in this way guarding against a long series of sequels such as organization of the exudate, shrinking of the lung, and compression of neighboring organs.

"As soon as the somewhat exaggerated zeal to remove serous exudates by means of puncture shall have abated, the surgical treatment of pleural exudates, performed according to strict indications, will rank as one of the highest achievements of our time,* as is shown by the various publications and numerous statistics of Tulschek, Kussmaul, Bartels, Ewald, Fräntzel, Oeri, König, and many others."

The above statements, which I made in Eulenburg's "Real-Encyklopädie," 2d edition, have been fully confirmed by the subsequent course of events. Since experience has taught us to estimate puncture followed by aspiration, which as a rule acts only symptomatically, at its true value, and strict indications for the evacuation of an exudate have been formulated, the results of operative treatment in all forms of effusions have become exceedingly satisfactory. The more we refrain from polyphragmasia and avoid useless puncture and frequent irrigations, the more favorable are the results obtained.

Whether the favorable results obtained in the operative treatment, especially of empyema, may not be partly due to the circumstance that the disposition of men for the so-called traumatic diseases has diminished during the past decades, with a corresponding rise in the disposition for certain internal diseases, such as tuberculosis, diphtheria, cardiac disease, etc., and a time may come when these brilliant results—which ought to be attributed less to the use of antisepsis than to that of asepsis, the method adopted by nature herself in effecting a cure—can no longer be obtained, it would be difficult to decide.

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* Half a century ago the operation was in ill repute. Even the well-known surgeon Du Puytren does not appear to have seen any good results following operative treatment of pleural exudates. It is said that when he himself was suffering from a bad case of pleurisy he refused operation, with the remark that he would rather die by the hand of God than by that of the operator. As every one knows, this illness had a fatal termination.

The history of medicine shows that what we regard as the result of treatment is always more or less the general product of factors whose influence is modified by anything that we may do only in a very slight degree, and in those rare cases in which we early recognize the direction and magnitude of these natural tendencies.*

1. MEDICINAL TREATMENT.

A certain advance in the medicinal treatment of pleurisy is found in the employment of the *salicylates*, which have been warmly recommended, especially by Aufrecht, Fiedler, Dock, and others. Recent cases, especially such as are accompanied by high fever, appear to be favorably influenced, according to my own experience, if they are treated early—if possible, at the very beginning of the disease—with large doses of sodium salicylate. On the other hand, the remedy is absolutely valueless in the torpid, anemic, and protracted forms. It is not efficient if given after the exudate has become stationary; or at best its favorable effect does not make itself felt as promptly as in other cases. The drug during the first few days must be given in doses of 4.0 (ʒj) to 8.0 (ʒij) per diem, the dose being reduced as improvement takes place.

A justification for the use of the salicylates in pleurisy is found in the investigations of Rosenbach [9] and Pohl, who found that the derivatives of salicylic acid after internal administration always find their way into the fluid contents of the pleura, be they exudative or dropsical, whereas the iodids appear only in transudates (due to stasis) and never in exudates, at least in doses that fall within therapeutic limits.

By this I do not mean to say that the *iodids*, which have a distinct antiphlogistic value, should be abandoned; I merely wish to define the special indications for the two remedies by pointing out that the salicylates are peculiarly adapted to recent cases, because they traverse the tissue-channels rapidly, while the iodids, which are arrested in the vessels or in the cells, exert a more intense local effect, and are, therefore, most to be recommended in cases of delayed resorption. In protracted cases and when the digestive disturbance or anemia are not pronounced, the preparations of iodin, including iodid of iron, exert a distinctly favorable influence. Even free iodin may possess some value. It has been especially recommended by G. I. C. Müller as a particularly effective agent in the form of the *tincture of iodin*, given in doses of 3 to 5 drops diluted in 50.0 (about f℥iss) of pure water.

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* See O. Rosenbach, "Die Entstehung und die hygienische Behandlung der Bleichsucht," Leipzig, 1893, pp. 6 *et seq.*

I should, therefore, recommend the use of iodids only in the later stages and in febrile cases, in doses of about $\frac{1}{2}$ to 1.0 gm. (gr. viij to xv) per diem. *Iodoform* (in pills of 0.02—gr. $\frac{1}{3}$—each) and *iodid of iron* (0.02 to 0.05—gr. $\frac{1}{3}$ to $\frac{3}{4}$) in combination with small doses of arsenious acid (1 to 2 mg.—gr. $\frac{1}{60}$ to $\frac{1}{30}$—given three or four times a day) or mineral waters containing arsenic (such as Roncegno, Levico, etc.), appear to be of some value in hastening resorption.

Digitalis in the form of the stronger infusion is recommended not as a diuretic, since it has a diuretic effect only in cardiac weakness and when vagus innervation is deficient, but as a heart tonic whenever the signs of this form of cardiac weakness due to paralysis of the vagus, particularly a small and frequent pulse, can be demonstrated. In the acute febrile stage an infusion of digitalis (1.5 : 150.0) may be given with great advantage.

The *caffein* preparations I prefer to reserve for the later stages where the diminution in the cardiac activity is not due to paralysis of the vagus, but to insufficient filling of the cavities on account of muscular weakness; that is to say, true myasthenia (not myatonia). In these conditions the double salts of caffein (caffein and sodium benzoate or salicylate) in solution or in powder three to four times a day (0.1 to 0.2—gr. $1\frac{1}{2}$ to 3) are appropriate. Subcutaneous injections of caffein (2.0 to 10.0 of distilled water), one Pravaz syringeful two to three times a day, have also been recommended.

The so-called diuretic remedies, with the exception of digitalis and caffein, which I do not include among true diuretics, in reality have no direct influence whatever on diuresis; and even if very large doses may appear to have such an influence, they would nevertheless have no effect whatever on the absorption of pleuritic exudates, since neither the excretion of exudates (not transudates) into the serous cavity nor their absorption has any recognizable causal connection with the excretion of water from the kidneys, intestine, or skin.

The stoppage in diuresis and diaphoresis which usually accompanies a large exudate is not due to any insufficiency (paralysis) of the kidneys or glands of the skin, but solely to the fact that, owing to the mechanical obstruction to the circulation, the excretory organs which in themselves are capable of working are not supplied with a sufficient amount of nutrition to enable them to work.

The fact that in severe cases of pleurisy the exudate continues to increase in spite of profuse sweating is a better proof than any theoretic considerations that the loss of water incident to sweat-production is not always necessarily inimical to the secretion of water in the pleural cavity, though it is true that in many cases the accumulation of an exudate is associated with striking dryness and feverishness of the skin.

Sweat secretion is a purely regulative process, associated with fever but bearing no recognizable relation to the formation of an exudate. In any event the amount of water in the body would have to be reduced to a minimum by unjustifiable measures before this hypothetic and impossible desiccation of the body would exert any influence on the processes going on in the inflamed pleural cavity, where the inflammatory irritants are greater than in any other part of the body and therefore lead to chemical combination and exudation of water.

The diuretics and digitalis owe their reputation of assisting the absorption of fluid effusions solely to their undeniable efficiency in all forms of stasis due to heart weakness, that is to say, any universal transudation; but we are not justified in regarding these conditions of mechanical stasis as equivalent with accumulations of fluid due to inflammatory processes, since the two are widely different, both in their physical and in their biologic relations.

I must, therefore, agree with Glax [6] when he says that the exudate does not diminish because diuresis is increased, but, rather, that the increase of diuresis in all cases is to be regarded as the expression of a beginning resorption which is characterized by elevation of the blood-pressure and increase in the amount of circulating blood.

Digitalis is unquestionably of value in protracted pleural effusions; only, however, when it can exert its effect as a heart tonic—that is to say, when the loss of absorbing power is solely due to cardiac weakness.

It is only by a careful consideration of the relations existing between the formation of an exudate and the chemical and mechanical work of the tissue that we can avoid therapeutic errors; for, except in the case of a purulent or putrid pleurisy, the exudate is not the primary cause of the irritation of the pleura, but the production of the exudate is the result of an abnormal irritant. Cure is not brought about by removing the fluid products of the inflammatory irritant, that is to say, the exudate; it occurs whenever the removal of the irritant makes a normal form of work possible, which does not, as in inflammation, consist merely in a quantitative and qualitative increase of the local (interstitial) tissue work (internal work), but also in the removal of the products of activity to other portions (by external work).

Under such circumstances diuretic remedies are no more able permanently to correct the abnormal conditions than direct evacuation of the exudate, unless the inflammatory irritant, the cause of the increased local work at the expense of intrinsic work, is eliminated.

It is not that diuresis is increased as the exudate diminishes, but the normal function of the kidneys, like the normal function of other organs, becomes re-established because the decrease in the exudate indicates that the endothelial cells have resumed their normal work; or, in other words, that the abnormal irritant has disappeared.

In **delayed resorption** and during **convalescence** a strong decoction of cinchona with hydrochloric or lactic acid, small doses of *quinin*, and an abundance of lemonade or buttermilk are to be recommended.

Antipyretic remedies when the fever is moderate seem to me useless, and treatment with sodium salicylate is recommended not because it incidentally exerts a material influence on the fever in favorable cases, but because it appears to exert a specific effect on the *causa morbi*, which in mild cases of pleuritis and articular rheumatism is probably one of the ordinary pus micro-organisms in a less virulent form.

Unless, therefore, the temperature reaches an alarming height, or, rather, unless the fever can be regarded as the only harmful agency, as is rarely the case, antipyretic remedies should not be used. If the gravity of the case is due to the intensity of the local phenomena, the fever, or, more correctly, the abnormally high temperature can only be influenced by very large doses of antipyretics with inevitable injury to the appetite and the patient's strength. Thus, the administration of antipyretics is very apt to be followed by collapse or profuse colliquative sweats. For this reason I do not advise the use of any antithermic remedies against a continuous fever, but prefer to give small doses of *quinin*, say, 5 cg. to 0.1 (gr. i to ij) three or four times a day, from the beginning. This has a tonic effect—that is to say, it appears to assist the body in adapting itself to the irritant—and reduces the temperature, or at all events does not disturb the natural course of reaction and accommodation.

During the later stages, if the fever is protracted, small doses of antipyrin or phenacetin may be given with advantage; the dose must be determined for every individual case, and should never exceed ½ gm. (gr. viij). It is best given about four to six hours before the temperature reaches its highest point, or, in other words, at the time of complete remission.

The greater the amount of sweating in this artificially produced apyrexia and the more abrupt the rise of temperature after the period of defervescence, the more certain is the conclusion that the dose was too high and that the forced antipyresis was uncalled for and hence injurious.

Bathing with cool water I believe to be contraindicated; warm baths and tepid sponging, while they have no perceptible influence on an obstinate temperature, stimulate the activity of the skin.

The *diaphoretic methods*, under whatever name they may be disguised, especially injections of pilocarpin, do not, so far as my experience goes, exert the slightest favorable influence on the course of the disease, nor do they even tend to hasten absorption. All such methods of treatment are to be condemned, if only because of the distress and weakening effect of profuse sweating which so many pleuritic patients suffer from. In fact, I think it is better to combat this form of paralysis of the sweat-glands by *cool sponging*, by the application of *ice-bags* to the abdomen and chest, and by small doses of *atropin* and *ergot*, since they almost always accompany a paralytic condition of the cutaneous vessels, or at least indicate a beginning impairment of the vascular system of the skin. Far from exerting any favorable influence on absorption, these paroxysms of perspiration only tend to undermine the patient's strength.

The disagreeable symptoms attending profuse perspiration can be temporarily relieved by the application of a *dusting-powder of* *salicylates,* which tends to check the sweat secretion, and by *sponging with vinegar.*

The well-known Schroth treatment or thirst treatment has now fallen into disuse everywhere, as it represents a mischievous interference in the economy of the body, and is unquestionably based on a mistaken conception of the connection between loss of fluids and the absorption of inflammatory exudates. Nevertheless Robinson has recently recommended another form of thirst treatment, consisting in the exhibition of salt, as if salt possessed the power of causing the watery constituents of the exudate to return to the blood channels by diosmosis.

The **bowels** must, of course, be regulated from the very beginning, because free catharsis, while it does not assist absorption, as is frequently thought, tends to diminish the dyspnea and sense of oppression. For this purpose the milder *salines, rhubarb* and *senna,* and, in the beginning of the disease, moderately large doses of *calomel,* are to be recommended; but one should never yield to the wishes of the patient, who is apt to attribute the sense of oppression and distressing pressure in the abdomen produced by the exudate to constipation, to the extent of relieving his sufferings by active purgation.

Osler [11] affirms that purgatives given in concentrated solutions (5 to 10 gm.—ʒj to ʒiij—of sodium sulphate in 30 to 60 gm.—f℥j to ij—warm water given before breakfast) possess the power of inducing the resorption of transudates and exudates by causing a profuse watery secretion into the intestine, a view which we believe is correct so far as transudates are concerned, but does not apply to exudates.

If the appetite is good and the fever is not excessive, there is no necessity to restrict the diet; in any case, however, it should be as easily digestible as possible and not too abundant (see page 1008).

The **general treatment** of the patient as regards hygiene must be carefully regulated from the beginning of the disease. As soon as the least suspicion of pleural disease exists, the patient should give up all work and go to bed. The room must be carefully ventilated, especially when there is profuse sweating and the air in the room is vitiated by the exhalations from the body. Free ventilation not only tends to eliminate the products of perspiration, but also exerts a certain beneficial influence on the reaction of the skin.

As soon as the continued fever has abated and morning remissions have made their appearance, the patient should be made to sit up at frequent intervals and go through systematic *respiratory movements,* so as to prevent the production of atelectasis. These exercises should always be carried out unless there is severe pain.

As soon as the fever has disappeared and the exudate begins to diminish, the patient may even be allowed to get up and move about the room for a short time; a little mild exercise materially facilitates absorption and is the best means of restoring the patient's strength in a comparatively short time.

Local antiphlogistic remedies as a rule do not affect the progress of a pleurisy to any great extent. They may, however, exert a favorable influence on the distressing pain and on the dyspnea if they succeed in diminishing the inflammation of the thoracic muscles or the hyperemia of the chest-wall. Thus, the parts may be painted with tincture of iodin or iodoform-collodion (2.0 : 30 collodion). Ichthyol in ointment or solution, or dry or wet cupping, blistering, mustard plasters, or ice may be resorted to.

The application of ice in acute diseases of the respiratory apparatus, especially in anemic persons, has not yielded very favorable results in my hands, and I would rather recommend the employment, in suitable cases, of moist heat by means of a Priessnitz compress, poultices, *cataplasma instantanea,* hot bottles, etc.

I have never seen any good results from strapping the chest with adhesive strips, as recommended by Perrin; on the other hand, Biedert [11] speaks favorably of this method, which he recommends especially for children. Otto [11] claims to remove the pain of pleurisy by compressing the thorax with a bandage 6 or 7 cm. broad, which limits the respiratory excursions. [Strapping, if properly done, has been of great service in our hands for the relief of pain.—Ed.]

In my opinion, the timely exhibition of *narcotics* offers the only successful means of relieving the pain of pleurisy and the spasmodic muscular movements due to the dyspnea. Small doses of opium or morphin, given by the mouth, or still better subcutaneously, at one blow remove the insomnia, the dyspnea, and the pain, and the relief thus obtained lasts many hours.

As regards **blood-letting,** it has been my experience that dry cups do not relieve the pain as well as wet cups. More radical methods of blood-letting are to be recommended only when the fever is very high and in robust plethoric individuals with a tendency to cyanosis, or when the pains are very severe and are only relieved for a very short time by morphin. In such cases very good results are often obtained by copious blood-letting by means of wet cups or leeches. Along with the pain the dyspnea and cyanosis disappear, sleep is induced, and the patient's spirits are revived.

In the future, unless all signs fail, blood-letting, especially venesection, is destined to resume an important place in the treatment of sthenic pleuritis.

2. OPERATIVE EVACUATION BY MEANS OF PUNCTURE.

The exaggerated ideas of the value of forced evacuation of pleural exudates by operative means appears to have been everywhere followed by a very salutary reaction. For a time, beguiled by the efficacy of the aspirator, we interfered at the wrong moment and in the wrong case, and did not observe due care in the amount of force used in evacuating the cavity. At present, however, there is a generally prevalent conviction that the operative evacuation of the exudate is a rational method only when applied to cases which are really—that is to say, after due consideration of individual peculiarities—suitable for operative intervention. All observers are, I believe, now agreed that puncture must be performed whenever the exudate is large or has existed for some time, or pulmonary edema is imminent, and that in the case of empyema free and permanent drainage for the pus must always be secured.

As regards other indications and the principles which determine the extent of evacuation, I shall repeat *in toto* the article on pleurisy which I published ten years ago in the section on pleurisy in Eulenburg's "Real-Encyklopädie," the correctness of which has been abundantly proved by subsequent experience. These indications and principles, so far as general rules can be laid down for conditions which are subject to such wide variations, afford a useful basis for therapeutic procedure.

It may be granted that evacuation performed with all the necessary precautions, both as regards antisepsis and the quantity of fluid removed, either puncture alone or puncture with mild aspiration, is in itself not only absolutely harmless, but even a necessary and useful surgical intervention, providing puncture is not followed by renewed accumulation of the exudate. The only possible argument against the necessity of the operation is based on those cases, which, it is true, are not altogether rare, in which an unexpectedly sudden reaccumulation of the exudate takes place. Something is gained even if puncture is followed by a small amount of reaccumulation, and the objection that this diminution might have taken place spontaneously is not tenable, since any hastening of recovery or diminution of the symptoms is to be regarded as a distinct therapeutic gain.

While it is true that puncture necessarily brings about a loss of albumin to the body, we do not even know whether in the case of a spontaneous absorption the albumin remains in the body and becomes available for purposes of nutrition, or whether, owing to the double demand on the organism and the increased work required for the complete decomposition and mechanical excretion of the inflammatory products, it is not eventually eliminated without being utilized for purposes of nutrition.

However that may be, the amount of nutritive material removed in moderate evacuation is inconsiderable, and besides it is not improbable that this material, after undergoing retrogressive metamorphosis, finds its way into the general circulation and there exerts a harmful influence, either by leading to embolism or thrombosis, or by setting up an inflammatory irritation of the excretory organs.

Hence the operation is a useful one whenever its effect on the general or local symptoms is such as to indicate that there has been a diminution or even abolition of the efficiency of the exudate-forming factors, if I may be permitted to use this expression. In such cases, instead of first resorting to the other doubtful methods of facilitating absorption, an attempt should be made to evacuate a moderate amount of the exudate; and although there is no specific sign indicating the approach of the most favorable moment, yet a diminution in the acutely inflammatory symptoms, the pain and fever, may be regarded as the signal of a return to normal conditions.

Even an advocate of the expectant method of treatment, who regards intervention at the improper moment as a technical error,—and I fully agree with this view, believing in the principle that operative delay in a case of serous exudate is less harmful than premature intervention,—ought to operate after the fever has completely disappeared or merely shows slight evening exacerbations, especially if, after exhausting every method of examination, it has been determined that the exduate has not increased for several days.

In most cases, I believe the indications need not be too restricted; for, by taking due account of individual conditions, a good therapeutic result may be obtained even if we overstep the limit just laid down. It should always be a rule, however, in all doubtful cases to withdraw no more than half of the amount that may be inferred by physical examination to be present. The removal of only a part of the fluid favors spontaneous resorption, and if in an unfavorable case the subsequent progress shows that the process of exudation had not reached its limit, such a discovery is not fraught with any very serious consequences to the patient.

In several cases I have performed puncture successfully after the height of the fever had passed and while there was still a moderate continuous fever, providing the exudate had not increased for several days. I always tap if the appetite reappears and there are no colliquative sweats, if there is no increase in the exudate, and the fever diminishes. In just such cases it will be found that the fever disappears immediately after operative evacuation of the exudate, and a favorable influence on the patient's general condition at once becomes manifest even although the exudate reaccumulates after a few days in diminished quantities. In such cases it may be assumed that the fever was not due to the primary inflammation which is the cause of the exudate, but that the exudate itself is to be regarded as the chief irritant of the pleura, which is especially sensitive and therefore not in the full possession of its absorptive faculties.

Accordingly, we find that exudates which owe their origin purely to a special local irritation or irritability—the foundation of all inflammatory processes—never show any distinctly favorable effect from operation if it is performed before the end of the inflammatory stage. As long as the conditions are abnormal, that is to say, as long as the inflammatory irritant is active, the exudate rapidly reaccumulates if operation is resorted to without due regard to special conditions.

There are, in addition, cases where puncture should be performed in the face of an increase of the exudate, since it is not altogether true that an increase in the exudate always depends on an exacerbation of the inflammatory process in the pleura; for it is to be remembered that when the activity of the tissues is disturbed, and the resorptive and mechanical powers of the protoplasm of the pleura are much diminished, the exudate itself may be a cause or even the only cause for a kind of transudation. In such cases, in which there is a combination of exudate and transudate, nothing but a mechanical evacuation of the fluid will remove the pressure from the tissue and enable the weakened resorptive power to reassert itself.

Exudates of this kind are, according to my observation, found mostly on the right side, and almost always associated with enlargement of the liver, as the effect of the pleuritic disease on the circulation manifests itself principally in the portal circulation. Only very large exudates belong to this class; exudates that do not reach to the middle of the scapula are usually not associated with transudation.

In regard to *pleuritis acutissima*, even in cases where the conversion of the serous into a purulent exudate which occurs very early has not taken place, I am inclined to indorse Aufrecht's[13] proposition of first resorting to aspiration, and, if this is followed by rapid reaccumulation of the exudate with high fever, at once to proceed to thoracotomy.

Even in a case of simple serous exudate in an adult, where the fluid had accumulated at an enormously rapid rate, and operative intervention was urgently indicated, I once performed thoracotomy without waiting for puncture and brought about an unexpectedly rapid and complete recovery.

In the case of **purulent exudates** tapping is never, in my opinion, to be recommended; and in spite of favorable results obtained by some authors, I cannot bring myself to recommend anything but early operation, for the chances of complete recovery are distinctly diminished by puncture, as I have only too often had occasion to observe.

Even in children I believe that resection or some other method of bringing about permanent evacuation of the pus should be aimed at, because the chances for complete recovery after resection are particularly good in children.

It is very difficult to lay down any general rules applicable to treatment in serous and hemorrhagic exudates occurring in carcinomatous, tuberculous, or cachectic individuals.

In cachexia, where the exudation is usually very slight and occurs only during the terminal stages, where dyspnea is due to loss of strength and not to local disturbance, no operative interference should be attempted, as the pain can just as well be relieved by morphin. In carcinomatosis, where the exudate is often quite considerable, puncture should be resorted to only if there is a vital indication.

On the other hand, I have often seen very favorable results follow puncture in isolated tubercular processes in the pleura and in pulmonary tuberculosis, although it is true that I have also seen such exudates undergo spontaneous absorption, and it is noteworthy that the tubercular manifestations in the lung often undergo a striking improvement during the existence of an exudate. In all cases of pronounced phthisis, therefore, even when dyspnea is great and there is continuous fever, I advise operation if it can be positively determined that the symptoms are due to the pleuritic exudate and not to the pulmonary process.

When there is any marked dislocation of neighboring organs, puncture is justifiable whether a serous or a hemorrhagic exudate be present; for there is always the possibility that the exudate itself is exerting some unfavorable influence on the movement of the blood, and thus directly laying the foundation for an increased mechanical transudation.

In all other cases, and when the exudate is moderate in amount and increasing slowly, I believe it is better to be conservative and not resort to puncture, for I have never seen any harm come to the patient through delay. If the exudate has remained stationary for some time, it is always better to tap.

Incidentally, it is to be mentioned that exudates that have become hemorrhagic during the act of puncture, either owing to the sudden change of pressure within the pleural cavity or to injury of the chest-wall, or to hemorrhage from a superficial focus in the lungs, are of course not included among true hemorrhagic exudates.

(a) **Mechanism in Evacuation of the Exudate by Puncture.**— In choosing the method of operative evacuation to be employed, whether aspiration, siphonage, or simple puncture, the most important consideration appears to be, *à priori*, the amount of pressure within the pleural cavity. The greater this pressure, the readier will be the spontaneous evacuation of fluid through the artificial opening, as may be seen in tapping the abdomen for ascites, where the contents are forced out in a steady stream if the pressure is high, or, to speak more correctly, if the tonus of the abdominal wall is such that the pressure is considerable in spite of the distention.

Now, it has been conclusively shown by manometric investigations that the pressure of an exudate within the pleura is comparatively low, and this pressure suffers an additional diminution during inspiration. In addition to this, the resistance encountered in the drainage-tube is comparatively great, because, owing to the relatively small width of the intercostal spaces and the danger incident to delayed closure of the puncture wound, we are in the habit of using trocars of rather small caliber, which by friction retard the flow of the exudate. As, in addition, the resistance due to friction in a pleural effusion, which contains morphologic elements and shreds of solid material and is thicker and of a higher specific gravity than ascites, is also relatively great, it occasionally happens, especially in exudates of long standing, where the tonus and contractility of the chest-wall and the expansibility of the lung have become impaired, that the fluid cannot be evacuated merely by inserting a trocar.

These conditions require a fuller explanation, as the fact that a copious flow may take place in spite of low pressure, while in very large exudates the rate of the flow does not correspond to the amount of the fluid, shows us at once that there are certain special factors which determine the pressure or rate of flow. In other words, the pressure exerted by the exudate, on which the rate of flow depends, is determined not by the physical distention of the chest-wall,—the degree of tension of the dead tissue between its points of fixation, but by a variable dynamic factor, namely, the variable tonus of the walls including the diaphragm, and by the intrabronchial pressure, which is equally variable because dependent on the tonus of the lung.

We know that every tissue possesses some degree of perfect elasticity, corresponding to its physical properties, by virtue of which it contracts after being distended, and, in addition, possesses a certain energy acting in the same direction, but effective only during life,—its *tonus*,—which can be diminished by certain minute irritants or stimuli and increased by certain other stimuli acting in the contrary direction. Any form of irritation of the pulmonary or costal pleura or of the lung itself alters the tonus of the chest-wall or of the lung itself, and therefore produces changes in the intrapleural and intrapulmonary pressure differing in kind and in degree from the changes which are dependent on the purely physical conditions; that is, on the elasticity residing in dead tissue and the external heat.

It is, therefore, to be remembered that the better the tone of the muscles, which are relaxed and not merely mechanically stretched by the action of the exudate, the higher will be the pressure of the exudate. If mechanical distention were the only factor, any marked though temporary increase in the tension would bring about irreparable dilatation of the walls, and the rapid and complete contraction of large cavities, which is observed in the majority of cases even after the evacuation of large exudates, would be utterly impossible.

To explain the possibility of living tissue being stretched beyond the limits of perfect elasticity a peculiar arrangement of the molecules must be assumed, permitting elongation of the tissue in the direction of traction (applied), not by increasing the distances between the molecules, —that is, by weakening their affinity,—but rather by the intercalation of other molecules which up to that point had remained passive. This arrangement prevents any disturbance of the intermolecular relations as long as the intercalation of new and efficient affinities is possible; that is to say, up to the point where true physical distention or separation of the molecules takes place. To recapitulate, we therefore assume that between two rows of molecules, possessing an affinity corresponding to that found in dead tissue or a slightly greater affinity, there is always another row of molecules possessing a slightly stronger affinity (living molecules) which during distention of the tissue step into the open spaces and thus maintain the same degree of elasticity that was present before distention of the tissue occurred. This condition, in contradistinction to physical distention, is termed *tonic relaxation.*

A hollow organ that has undergone this kind of relaxation is capable of largely increasing its volume without its walls becoming distended in the physical sense, and after evacuation of its contents can therefore retract without the action of elasticity, which of course presupposes a former distention.

Whenever the thoracic muscles and the diaphragm contract and dilate the thorax, the lung relaxes in the sense of the above definition, while its tonus is diminished; it is not distended by the inrushing air. When the traction which has caused the dilatation of the thorax ceases, the tonus of the lung at once reasserts itself and brings about retraction, the muscles meanwhile relaxing and allowing the ribs to become approximated.

The tonus of the thoracic wall is therefore not identical with the tonic contraction of the respiratory muscles; the latter effect dilatation of the thoracic cavity, whereas the tonus of the lungs and its membranes (the pleura) and a portion of the intercostal muscles effects contraction of the thoracic cavity. The tonic function of the bony wall of the thorax, including the muscles acting during inspiration, acts in direct opposition to the tonic apparatus of the lung.

What is the state of affairs when there is an exudate? According as it tends to excite or inhibit tonus, as has been explained on page 916, a change occurs in the tonus of the lung and of the thoracic muscles. As a rule, the tonus of the lung is increased; that is to say, the so-called intrapulmonary pressure, or, more correctly, the force of the air rushing into the lung, is diminished, while at the same time, owing to the action of the costal pleura, the tonus of the chest-wall is sufficiently diminished to allow the layers of the pleura to become separated and form a pleural cavity for the reception of the exudate.

Since, therefore, there is no distention in the physical sense, the pressure on the surface of the exudate is no greater than the pressure in the so-called normal pleural cavity, that is, in the capillary space between the layers of the pleura. Strictly speaking, it is zero, because the lung and the walls of the thorax, including the diaphragm, so long as the tonus is not affected, tend to contract concentrically or move toward the hilus.

If, however, both the lung and the chest-wall shrink in the same degree, there can be no movement of the exudate toward the opening in the thorax, since the pressure within the bronchi is always less than the atmospheric pressure which acts on the chest-wall. There is an undulation of the exudate, the waves during expiration traveling toward the lung and during inspiration toward the chest-wall; and when the respiratory movements and the vacillations of the tonus have ceased, the fluid remains immovable.

The pressure becomes positive whenever the contraction of the lung is less than the diminution in tension of the thoracic wall, and this condition of affairs occurs in the great majority of cases because the action of the voluntary muscles of respiration, especially the diaphragm, which dilate the thoracic wall, and therefore by their contraction diminish the tonus, is influenced by pain or other inhibitory stimuli. The greater the relaxation of these muscles, the greater the antagonistic activity of the tono-motors of the thoracic wall; and the smaller the inspiratory excursion,—if the tonus is maintained,—the greater the tension of the exudate.

The diminution in the respiratory surface brought about by the exudate sometimes produces a compensatory increase in the innervation of inspiration, which, if the tonus of the lung does not undergo a corresponding relaxation, can only lead to further enlargement of the pleural cavity and increase of the exudate.

Hence there is always some pressure on the surface of the exudate and a certain slight tendency of the lung toward the chest-wall. The greater this pressure, the better the chances for restitution, as the pressure guarantees a continuance of an effective tonus, which alone can bring about compensation; that is, closure of the pleural cavity.

Permanent pressure, permitting the fluid to escape during puncture, can therefore only be brought about by the pressure maintaining itself at, or even above, the former level by the action of the tonus and in proportion to the possibility of escape of the fluid. A necessary condition for the complete and uninterrupted evacuation of the fluid is the existence of a certain active power of diminishing the size of the cavity by contraction of the muscular wall and expansion of the lung.

What is the effect of making an opening? It reduces the pressure at the point of puncture to that of the atmosphere and at once permits the escape of an amount of fluid corresponding to the positive excess of fluid in the pleural cavity; that is to say, if the walls were distended in the physical sense, the amount of fluid evacuated would correspond to the degree of elasticity necessary to bring about a condition of equilibrium. The amount of fluid thus evacuated would therefore, in view of the slight although perfect elasticity, be inconsiderable, as, indeed, is found to be the case, whereas if the tonus—that is, a certain degree of energy varying in individual cases—is brought into action, the degree of pressure-difference or work performed may be quite considerable.

Furthermore, it is readily seen that for any effect to be produced on the exudate, the lung must be distensible; that is to say, capable of complete relaxation so that the intrapulmonary air-pressure may assist the action of the tonus of the chest-wall.

Complete evacuation can, therefore, take place only when there is, proportionate to the outflow, a diminution of the stimulus which maintains the tonus, allowing the lungs to retract and the voluntary muscles of respiration to perform that part of their work which increases the intrapulmonary air-pressure without destroying the tonus of the chest-wall. In a word, puncture will be followed by a stronger flow in proportion as the tonic innervation, which regulates the contraction and relaxation of the various regions, is preserved; for in that case there will be a moderate but uniform permanent pressure on the surface of the fluid, which, although it is low at any given moment, yet from the fact that it depends on considerable retraction of the wall and corresponding distention of the lung, represents a considerable absolute amount of energy.

It follows therefore that, paradoxical as it may sound, the pressure is not as low as our methods indicate, because we are not dealing with hydrostatic pressure or the effect of elasticity on dead tissue. It is only in such tissues that the amount of dislocation depends on the pressure of the fluid or the measurable forces at work during distention, that is to say, external influences; and this dislocation is proportional to the strength of these external forces. In a living and properly functionating tissue, on the other hand, movement is determined by the reflex tonic contraction of the cavity, that is to say, by internal forces which in the strictest sense represent a variable function of the nervous stimuli. For this reason the rate of flow remains the same long after the contents have become diminished and the level of the fluid has fallen, because the accelerating force remains the same or even becomes increased as the cavity continues to contract.

The paradox that a thorough evacuation takes place even when the amount of exudate is small is therefore easily explained if we remember that the tonic innervation, which is under reflex control, liberates very great amounts of energy, the successive development of which constantly tends to diminish the volume of the cavity and thus maintains a constant pressure.

Thus it is also seen why the manometer does not register the actual or possible amount of work done, but merely a relatively stable condition of equilibrium,—the temporary pressure on the surface, the magnitude of which is directly dependent on the resistance represented by the manometer, and which inhibits the tonus of the chest-wall. As soon as a manometer is inserted, there is produced a new condition of equilibrium which is directly due to occlusion of the cannula and lasts as long as the cannula remains occluded, manifesting but slight variations. It is only when a free outlet for the fluid is afforded that the walls are able to develop their full energy; that is, to utilize the forces at their disposal for the constant diminution of the cavity until a mean volume has been reached.

If, however, anything occurs to arrest the outflow, the walls, being under tonic influence, immediately adapt themselves to the contents. The surface pressure represents merely the condition of equilibrium corresponding to the intensity of the inhibitory stimuli, and not the highest possible amount of energy available for the expulsion of the fluid, just as a piece of iron does not show the same weight when it is placed on the scales—that is, under ordinary conditions—as when it is under the dynamic influence of a magnet.

If a manometer is connected with a trocar inserted into the interior of the pleural cavity, and the resistance to the outflow is thus increased by a certain amount corresponding to the increased resistance offered by a column of mercury or other fluid, or if the outlet is obstructed altogether, the activity of the reflex apparatus will at once suffer a slight diminution until a new state of equilibrium is established.

Hence the column of mercury in the manometer indicates merely the state of equilibrium, and not the forces available for the expulsion of the fluid when there is a free outlet. In other words, the force available for the expulsion of masses in the living body at once becomes diminished if the outflow is obstructed, and the most unstable equilibrium is converted into a less unstable or even into a stable equilibrium.

The evacuation of a fluid from a cavity formed by living tissues is, therefore, dependent on the pressure within the tissue,—that is, on the tonus,—and not on the pressure at the surface of the exudate measured under certain permanent conditions of resistance, because the change in pressure which, owing to the free communication established between the thoracic cavity and the atmosphere, occurs at the site of the puncture may liberate an amount of tonic energy which cannot be calculated and persists for some time.

Corresponding to these variations in the tonus of the wall, the fluid follows the line of least (tonic) resistance and gravitates toward the puncture wound, and this will continue as long as the outflow itself acts as a stimulus for the continued approximation of the tissues or as a foundation for the new tonic innervation which produces a diminution of the cavity. Under the most favorable conditions—that is to say, when the expansion of the lung is sufficient and the tonicity is greatly increased (intramuscular contraction of the wall)—the evacuation may continue until the size of the thorax approaches the mean volume at the end of an ordinary expiration under normal conditions.

A uniform outflow can take place only when the original pressure at the surface is maintained by an increase in the tonic pressure of the thoracic walls and diaphragm or by a diminution of the pulmonary tonus which is identical with an increase of the intrapulmonary pressure; or, in other words, expansion of the lung.

Hence the circumference of the thorax must diminish in proportion to the diminution of the exudate until this concentric contraction reflexly sets the voluntary muscles of respiration into motion. As soon as respiration begins, the uniformity of the flow can be maintained only if the respiratory movements as such do not exert any material influence on the intrapleural pressure; or, in other words, if, during the inspiratory dilatation of the thorax, the pressure on the fluid, its movement toward the opening, is not less than during expiratory contraction. These conditions can, of course, be fulfilled only if the lung at each inspiration expands at least as much as the thorax, and the thorax during expiration contracts to the same extent as the lung. Granting that there is a possibility of respiratory movements being performed, an inspiratory diminution and expiratory increase in the flow will take place, unless the inspiratory change of volume is compensated for by the entrance of sufficient air into some portion of the lung, while the resistance to the expiratory contraction of the lung, which is represented by the interbronchial pressure, is greater than the resistance at the opening in the chest-wall. The flow will be completely arrested if during expiration the intrapulmonary pressure, or, to speak more correctly, the stimulus exerted on the pleural surface of the pulmonary pleura which effects the contraction of the lung, becomes as great as the pressure on the pleural fluid through the outer wall of the thorax.

Hence a positive pressure—that is, one which permits a flow toward the external air or through the outer thoracic wall—can exist in the pleural cavity only if the relaxation of the tonus of the lung—its power of expansion—is greater than the power of expansion of the thoracic wall. This result is partly attained by the necessity on the part of the thoracic wall during expansion to raise the contained fluid, which is, of course, more difficult to move than is the elastic lung tissue. The greater the inspiratory relaxation of the intrapulmonary tonus and the greater the expansion of the lung, the greater will be the pressure exerted during inspiration on the exudate from the lung toward the opening in the chest-wall.

If the lung were to follow the contraction of the chest-wall uniformly, the fluid could not escape, because the inspiratory enlargement of the thoracic space would be constantly filled by the lung, whereas the first condition for the evacuation of an exudate is that the expansion of the lung be greater, or, rather, that the rapidity with which it moves upon the exudate be greater than the rapidity with which the thoracic wall retracts. If the distention of the thoracic wall is more rapid than that of the lung, a stoppage of the outflow will occur at each inspiration; while during expiration, when the weight of the thoracic wall reinforces the hydrostatic pressure of the fluid, the interference is less, until finally both parts move with the same rapidity and the flow is completely arrested.

The mere hydrostatic pressure, therefore, plays a very inconsiderable part, as we generally do not have to deal with large columns of fluid. Even if puncture is performed in the dorsal position, when the level of the fluid is at its lowest, no difference is observed in the rate of flow. The most important factors in the regulation of the flow are the peculiar construction of the pleural cavities, which may in a sense be compared to the effect of capillarity, and the tonus of the tissues, that is, the dynamic power which distinguishes living tissue from a similarly constituted dead tissue, just as the machine differs from the aggregate of raw material.

Elasticity is not the only efficient factor in the evacuation; another of equal importance, and one that works in the same direction, is the tonus of the tissues. All tissues, especially the hollow muscles, not only possess the power of expanding, under the influence of an internal or external distending force, in proportion to the amount of this force and of contracting after the force has been removed, but also possess the power, by a mere alteration in their innervation,—that is, under the influence of minute stimuli,—of developing energies available for diminishing their volume which are not possessed by dead tissue.

In this way considerable masses of matter are taken up and carried off without compression by corresponding external forces. By the exercise of mere intermolecular force and in response to a slight stimulus the volume is diminished or augmented. It is, therefore, less a static traction or pressure due to stretching of the tissues, than a dynamic factor, capable of various degrees of work in accordance with the stimulus—in other words, the tonus—that is at work. And, in contradistinction to what we usually understand by static process, the possibility of movement is greater, the less the tissues have, under the influence of great external forces, suffered a true distention; that is to say, the smaller the original volume of all the parts as determined by their tonus.

The greater the mean tonus, the greater is the volume and capacity for work of a hollow organ like the lungs and the heart. The more atonic the tissue in a biologic sense, the nearer it approaches the physical state of equilibrium, and the less, therefore, the possibility of efficient active change in volume which is the basis of all external visible work.

The tissue of an emphysematous lung and of an atonic heart are susceptible of physical distention by external forces. The volume is increased when heavier weights are applied, and contracts to a corresponding degree after the weight is removed. On the other hand, it is not susceptible to the increased influence of the inner (intermolecular) forces which form the basis of the tonus and of the vital process. When, therefore, the power inherent in the molecules of living tissue of attracting and repelling one another more actively within a certain region (organ) is lost or diminished (when the tissue dies), the tissue thus affected even during life tends to undergo a corresponding augmentation in volume; but this, I may again emphasize, is not due to elasticity under the influence of external distending forces, but merely to a loss of the greater, *vital* power of attraction possessed by the molecules.

Atony, that is to say, loss of the special power of attraction and repulsion, which forms the foundation of vital processes, is therefore not identical with distention, which is effected purely by external forces; nor is atony identical with relative enlargement of the volume of an organ due to the loss of an especially active tonus, an enlargement which not only produces a greater capacity, but also, on account of the accompanying increase in the active energy necessary for contraction, becomes the foundation of any increase in work.

In the heart I have called this condition hyperdiastole,* in the lung it corresponds to *volumen pulmonum auctum inspiratorium* (inspiratory increase of the pulmonary volume). Emphysema or its precursor, *volumen pulmonum auctum permanens* (permanent increase of pulmonary volume), is comparable to atonic, absolute or relative, dilatation. In the former case the alteration is not due to the functional processes,—to changes in force-production,—but to visible and in part structural changes, the especially active molecules of the essential tissue being replaced by the less active molecules of connective tissue (degeneration and atrophy of the parenchyma).

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* O. Rosenbach, "Die Krankheiten des Herzens und ihre Behandlung," Wien, 1893.

In emphysema and in permanent dilatation of the heart the tissue is, therefore, not overstretched in the physical sense, nor is it to be regarded as relaxed in the biologic sense, since relaxation means merely a temporary and not a permanent loss of tonus. It is, in fact, atonic and asthenic, that is to say, its molecules permanently lose their special power of attracting and repelling one another, or, in other words, their capability for the greatest degree of intermolecular work which characterizes the organism. In normal and excessive diastole and inspiration, on the other hand, the tissue becomes periodically relaxed by a loss of tonus, but not distended (hypertonia).

In yielding tissues such as the normal thoracic walls, which by virtue of the strength of their mean tonus are capable of considerable augmentation of volume when the innervation is relaxed, that is to say, can relax without becoming distended,—in a word, in thoracic walls of normal elasticity, founded on perfect tonus,—the capacity of the thoracic cavity, and hence the power of regaining its volume after it has been diminished, is especially great. In youthful individuals the pleural cavity accordingly holds comparatively large masses of fluid without becoming markedly distended, or losing its power of contraction, as it would if the dilatation were to last for some time. When an opening is made in the thorax, the tonus is at once restored and the contents are completely evacuated, so that *restitutio in integrum* appears comparatively early.

To recapitulate once more, the enlargement of the thoracic cavity may occur in one of two ways: namely, by a reflex relaxation of the tonus of the chest-wall, as during the accumulation of an exudate; or, as in emphysema, by an inspiration (maximum contraction of the voluntary muscles) which presupposes abolition of the tonus of the chest-wall and of the lung; since the latter favors contraction, or, in other words, expiration.

During the inflammatory stage of an exudate anything which tends to excite inspiration is therefore harmful; hence forced contraction during aspiration is to be avoided, since it acts as a maximum stimulus to the inspiratory muscles. It does not to the same extent remove the inhibiting stimuli which resist the expansion of the lungs, and certainly does not tend to restore the expanding power of the lungs which has been lost through local inflammatory and other influences.

Very little force should therefore be used during evacuation.

Under such circumstances a continuous flow or the late appearance of respiratory changes in the flow (stoppage during inspiration) are favorable prognostic signs, while a sluggish flow and marked respiratory changes in the flow occurring early indicate insufficiency of tonus and disturbance of the relation between the expanding power of the lung and the enlargement of the thoracic cavity by the voluntary inspiratory muscles. These phenomena also prove that restitution after evacuation of an exudate is brought about not merely by the forces of elasticity, but principally by the action of the normal tonus.

The tonus of the lung in general works in a contrary direction to that of the chest-wall, which, by fixing the layers of the pleura within a large cavity, tends to favor the expansion of the organ. To put it more precisely, the tonic innervation of the lung is antagonistic to that of the chest-wall. The vagus which controls the tonus of the inspiratory thoracic muscles also transmits inhibitory impulses to the tonus of the lungs, and vice versa. While the lung is contracting under the influence of its tonus, the chest-wall is relaxing, and, conversely, while the lung is relaxing, the inspiratory muscles are beginning to contract, that is to say, are exhibiting the greatest degree of tonus. The contraction of the voluntary muscles of inspiration is identical with the greatest degree of dilatability, the maximum relaxation, of all other thoracic structures, so that in reality, when the various portions of the pulmonary system, which are under reflex influence, are working together under normal circumstances, the tonus of all the structures which form the true respiratory apparatus (the lungs, the pleural sacs, and the constrictors of the intercostal spaces) at the same time increases and diminishes, while the opposite is true of the tonic (contractile) innervation of the voluntary dilators of the thorax. Inspiratory contraction of the muscles must be preceded or accompanied by a decrease in the tonus of the lungs and of the chest-wall; the expiratory relaxation of the former must be accompanied by a corresponding increase in the tonus of the latter.

This short description of the mechanism of normal respiratory movements and the same movements under the influence of an exudate we believe affords a better explanation of the somewhat complex processes which enter into the question than could be given by merely taking into consideration the purely static relations; that is, the influence of forces in equilibrium. If one were to disregard dynamic influences in the attempt to explain the peculiar and exact mechanism of respiration, which in warm-blooded animals is controlled by the vagus, many phenomena would remain unexplained. The action of this nerve especially, which is the agent of tonic innervation, would remain shrouded in obscurity.

As a rule, it is therefore not necessary, especially in recent exudates, to use any aspirating apparatus or to employ any method requiring great rarefaction of the air, because the proper working of aspiratory evacuation depends on the power of the lung to expand, and this advantage is lost if the tissues affected by the morbid inflammatory irritation of the pleura, the lungs, the diaphragm, and the chest-wall, are incapable of relaxing or increasing their tonus to a degree corresponding with the strength of the aspiration used.

If the irritation of the pulmonary pleura is so great that the lung is forced by its tonus to contract more forcibly than usual, forced aspiration merely gives the opportunity for renewed exudation. For, since, in spite of evacuation of the fluid, the inflammatory irritation, which is partly inherent in the pleura and incites contraction, as well as the effort of the lung to contract under the influence of this irritant, persist, further opportunity is afforded for a special diminution of internal (intrapleural) pressure resulting in the production of a still larger capillary space either in spite of, or because of, the increased inspiratory activity of the thoracic muscles induced by the excessive aspiration. As the capillary space must be filled whether the lung is capable of expanding or not, a renewed transudation or exudation may become necessary.

Hence it does not take a teleologist to see in the painful expiratory movements and in the occurrence of cough and pulmonary edema an effort to bring about rapid compensation for the sudden and forcible removal of pressure from the pleural cavity; or, in other words, to diminish the excessive distention of the lung.

Accordingly, very little force should be used in aspiration, and, in contradiction to the views expressed by myself nearly twenty years ago, when I placed too much reliance on the purely mechanical explanation of the process, I would recommend **tapping without aspiration** or only with the help of **siphonage,** that is to say, using only enough pumping action to overcome the resistance of the friction in the cannula.

It is always possible to connect this siphoning apparatus—if it does not work properly because the flow is too great or because of other obstacles to the flow, such as coagula, shreds of tissue, etc.—with another apparatus admitting of greater aspiratory force.

In order to avoid or at least to minimize these disturbing factors, I would recommend the use of a large cannula at the outset. The increased rapidity of the flow afforded by a cannula of larger caliber more than compensates for the slight excess of pain inflicted by the introduction of a larger instrument. When the exudate is thick and has existed for some time, the thickest trocar that can be conveniently introduced between two ribs should be employed.

(b) **Aspirating Apparatus.**—The aspirating apparatus of Mosler-Peiper [12] consists either of voluminous syringes or of large receptacles in which the air can be rarefied at will, as in an air-pump, by means of a syringe provided with valves. Those in commonest use to-day are Dieulafoy's aspirator and Potain's apparatus (Rasmussen's bottle), both of which are very exact in their results and easy to manage. In many if not in most cases evacuation can be effected by means of a simple fountain syringe (a funnel and rubber tube connected to a trocar), which is first filled with an antiseptic solution and then immersed in a vessel containing a similar solution (Ewald,[12] Risel,[12] Goltdammer,[12] and others). Pleuritic exudates can also be aspirated by means of a rubber ball (Rosenbach [9] and Unverricht).

Fürbringer,[12] starting out with the assumption that in certain cases of pleurisy a stronger degree of aspiration is indispensable and a simple fountain syringe does not always suffice, constructed an apparatus which was intended to combine the advantages of a fountain syringe with the necessary degree of aspiration, which is susceptible of accurate measurement. In simple cases I have made a kind of fountain syringe with the rubber tubes from Potain's apparatus, and when a stoppage occurred in the flow, connected it with a vacuum bottle, which can be done in a moment and without any difficulty.

Revilliod claims to have used a rubber siphon with a rubber ball in its continuity to some advantage in the treatment of purulent pleurisy.

Judging from personal experience, I do not think it is very important whether the capillary trocar or the aspirating needle is used, although the latter is said to be capable of producing dangerous complications by injuring the lung or the diaphragm if the patient is restless. Although this is a mere theoretic possibility, and thousands of punctures have been performed in practice with the aspirating needle without any bad results, it is always better to choose a trocar for the operation, on account of the possibility of removing any obstruction in the cannula, such as fibrin-clots or shreds of pus, by the simple procedure of inserting the stylet. Potain's and Fräntzel's trocar[12] and Kahler's[12] instrument are excellent contrivances which answer every requirement because they are easy to handle, and with its use the possibility of entrance of air into the pleural sac is reduced to a minimum. Kahler's instrument consists of a small aspirating syringe, which, instead of the ordinary needle, carries a so-called capillary trocar-cannula possessing a lateral opening closed with an air-tight stop-cock and a stylet which is connected with the piston of the syringe. Another good instrument is Fiedler's double trocar, a hollow needle with a movable cannula inside which can be thrust forward after the puncture has been made and thus renders the point of the instrument harmless.

(c) **Method of Performing Puncture.**—In performing puncture cerebral anemia will best be avoided by placing the patient in the *dorsal position* with the shoulders slightly raised, or in an oblique *(lateral)* position with the affected side somewhat higher than the healthy side. The sitting posture is less desirable. It is an error to suppose that the sitting posture is preferable because the fluid in that position gravitates to the bottom, and thus facilitates complete evacuation, since the possibility of evacuation depends less on the hydrostatic pressure of the column of fluid than on the pressure and tension in the lungs and thoracic walls. Besides, the erect position involves certain disadvantages, such as increased muscular exertion, especially of the diaphragm, and diminished blood-supply to the brain. The coughing paroxysms which often disturb the operation are less apt to occur in the dorsal than in the erect posture.

If the flow becomes arrested, a slight change in the direction of the cannula or in the patient's position, or asking the patient to cough once or twice, will suffice to re-establish the flow. It is always a good plan to interrupt the flow from time to time by closing the stop-cock.

As regards the *point of insertion* of the needle, the region between the median and anterior axillary lines in the fifth, sixth, or seventh intercostal space on the left, or the sixth and seventh on the right side is recommended. If this area is chosen, there will be very little danger of wounding other organs, especially the diaphragm. In any case, except when there is a very large exudate, it is best not to make the puncture too near the lower border of the lungs, as, owing to the rapid rise of the diaphragm, the cannula becomes considerably displaced as soon as a small quantity of the exudate has been evacuated.

Puncture can also be made in the *back*, particularly when the exudate, on account of adhesions, does not extend far beyond the posterior axillary line in front, and is bounded by an abruptly descending line in the lateral wall of the thorax. In such cases we are forced to operate with the patient in the *sitting posture*, because the oblique position usually interferes too much with the expansion of the healthy lung. The patient should be supported by an assistant and should be given a stimulant during the operation.

The same **precautions** are to be observed in puncturing the thorax as in opening any other part of the body. The instruments must be carefully cleansed and examined to see that they are in proper working order, particular care being had that the stop-cocks are properly closed. The skin should first be carefully cleansed and the instrument inserted in the middle of the intercostal space so as to avoid the ribs.

If, as never ought to happen, the trocar strikes against the rib, it should at once be withdrawn and inserted in another place; for, although it is possible to insert the instrument obliquely into the intercostal space by gliding past the lower border of the rib, the procedure gives the patient unnecessary pain, and, on account of the oblique course of the cannula, interferes with the flow of the exudate.

Sometimes a costal pleura offers so much resistance that the operator thinks he has struck a rib, but on again inserting the instrument the condition of affairs is at once explained if the same difficulty is encountered; greater pressure will then be required and the instrument will pass into the pleural cavity without any difficulty. After a little practice the operator will learn to distinguish between the peculiar resistance presented by the pleura or a tensely stretched muscle and the resistance of a bone, and, instead of withdrawing the trocar after the first insertion, will simply exert extra force to push it through the thickened pleura.

The operation should be performed rapidly, the depth to which the instrument is to be inserted being gaged by placing the index-finger against the needle after the thickness of the thoracic wall has been estimated. It is also necessary to press the outer end of the trocar firmly against the flat of the hand so as to prevent the point from being thrust into the cannula when the puncture is made.

Local anesthesia is unnecessary if the operation is performed so rapidly as to avoid all unnecessary movements and convulsive contractions.

After the cannula has been inserted, the operator, before proceeding with the evacuation, should satisfy himself that it is freely movable. The cannula should be held during the entire time of the evacuation so as to spare the patient any unnecessary pain and prevent its slipping out of place. It also enables the operator to satisfy himself at every moment that the cannula is freely movable and to determine its position in the fluid or to discover any collision with the diaphragm or the lung. Toward the end of the operation the outer portion of the trocar should be gradually raised so as to keep the inner opening as far as possible below the level of the fluid.

If anything occurs to stop the flow, the stop-cock should be closed and the direction of the cannula slightly altered; any attempt to overcome such an obstacle by forced aspiration is a grave technical error, as it may easily lead to laceration of the pulmonary tissue.

Evacuation may be continued as long as the fluid continues to flow readily and there are no symptoms of collapse, no excessive cough, cerebral anemia, or palpitation of the heart. I have rarely evacuated more than 1500 c.c., though of course there are cases where greater quantities can be evacuated without any difficulty. As soon as the patient begins to cough, especially if he expectorates a frothy sputum, I always stop the evacuation either temporarily or permanently.

It is well to give the patient small quantities of *antispasmodics* during the puncture.

When the evacuation is deemed sufficient, the trocar must be removed as rapidly as possible after giving the patient due warning; an unexpected withdrawal tends to frighten him. The small wound is then closed with adhesive plaster or collodion. A slight oozing of fluid occurs very rarely and does no harm. The entrance of a small amount of air into the pleural sac is of no importance, and not rarely occurs in spite of all precautions if the assistants are somewhat careless or the rubber tubes are torn loose from the joints.

In using the aspirating bottle it is important to see that the syringe is properly put on, so as to avoid compressing the air in the bottle instead of rarefying it with the first strokes of the piston. Before proceeding with the operation it is therefore well to see that the proper valve of the syringe communicates with the bottle and that all the connections are firmly in position.

To avoid too rapid evacuation of very large exudates Lewaschew[1] [2] has proposed the following procedure. He evacuates only a portion of the exudate and immediately introduces a sterile 0.7% salt solution; this he again evacuates, and repeats this process until the exudate is practically replaced by normal salt solution, which is said to be easily and completely absorbed even when the inflammation is recent. For my part, I strongly advise against any such procedure, as I have found that the introduction of sterilized solutions tends to increase the irritation of the pleura and furthers suppuration or putrefaction. Nor can I see any advantage in substituting normal salt solution for a serous exudate in cases where the endothelium is not capable of absorption, or where the hyperemia produced by a maximum irritation lays impossible demands on the activity of the tissue.

The **after-treatment** obviously depends entirely on the result of the operative intervention. If the exudate rapidly reaccumulates and is attended by a marked rise of temperature, the patient must be treated according to the above-mentioned therapeutic principles. If there is no renewal of the exudate and the other symptoms improve, systematic *breathing exercises*, *outdoor exercise*, a *strengthening diet*, and *hydrotherapeutic measures* should be employed as soon as possible. Such cases are greatly improved, if the season of the year permits, by a sojourn in appropriate health resorts or at the seashore.

(d) Unfavorable Accidents during and after Evacuation.—By observing the above-mentioned precautions it is usually possible to avoid the occurrence of stenocardial attacks, edema of the lungs, and large hemorrhages from deposits in the pleural cavity or superficial foci in the lung, which are caused usually by too rapid removal of pressure from the surface of the lung, and only in rare cases by brittleness of the vessels in the pleural deposits. When, however, profound changes in the lungs, such as gangrenous foci, cavities, and emphysema, are present, even the most scrupulous care will not always succeed in avoiding rupture of the tissue.

It is not always possible to oversee and guard against the possibility of *congestive and edematous conditions* being produced in the tissue of the lungs by the sudden hyperemia of the blood-vessel following evacuation, especially when the lungs have long been in a condition of atelectasis; but if evacuation is carefully performed and the first symptoms of congestion are attended to, these unpleasant consequences can be reduced to a minimum.

Especial care is required to prevent the development of distressing *cough* with expectoration of tough, frothy mucus accompanied by a kind of collapse. Severe cough is a positive sign of marked hyperemia, while only slight attacks of coughing without expectoration or dyspnea merely indicate that the lung has become completely filled with air. A certain degree of inflation of the lung acts as a strong expiratory stimulus, that is to say, it liberates nervous impulses which, as Hering and Breuer have shown by their well-known experiments, favor the contraction and decongestion of the lungs, or, in other words, give rise to forced expiratory movements and attacks of cough. Such paroxysms of cough therefore indicate that the pressure has been sufficiently released from the lung and that the lung tissue is unable to respond to the excessive inspiratory stimuli by expanding in an equal degree, but is forced to undergo contraction by means of expiratory movements. A moderate amount of coughing may safely be disregarded, as it merely contributes, by raising the intrabronchial pressure, to the expansion of the lungs; if it becomes troublesome to the patient, it can always be controlled by hypodermic injections of small doses of morphin. Attacks of greater intensity and duration, unless they occur in unusually irritable individuals, should always be regarded as the signal and precursor of active congestive hyperemia and edema of the lungs. It is a symptom of such alarming gravity that the operation should immediately be terminated, particularly when it manifests itself after a considerable amount of exudate has already been evacuated.

The symptoms of *hyperemia* and *edema* may occur in the healthy or in the diseased lung, or in both lungs, and may lead to a fatal termination with increasing dyspnea and cardiac weakness.

The so-called *albuminous expectoration* after thoracocentesis, first described by French authors (Terrillon,[7] Besnier,[7] and others) and later by English (Johnson, Duffy) and German observers (Scriba[7]), has a certain symptomologic value as a sign of pulmonary congestion. It indicates the development of edema after the sudden release of a compressed lung, and is attributed by Leichtenstern, on the strength of Cohnheim's investigations, to a nutritive disturbance in the walls of the vessels secondary to long-continued interference with the blood-supply.

As may be seen by what has been said before on this subject, we do not believe that the condition is due to a true nutritive disturbance of the vessel-wall, as it seems to us that, owing to the maximum tonic relaxation—not distention—of the vessels which suddenly receive an enormous amount of blood, the interstices of the tissues, which under normal conditions of tonus resist the passage of intact blood-corpuscles and possibly even of molecules of serum, suddenly open and thus bring about a flooding of the true parenchyma cells from the minute lymph-paths by which they are surrounded.

The maximum relaxation of the blood-vessels is of course accompanied by a maximum amount of work on the part of the protoplasm filling these lymph-spaces to their utmost capacity. This increase of the internal local work necessarily means a reduction in the work of transportation.

If fluid is passed through a moderately compressed sponge, the movement within the sponge will at once be retarded by relaxing the compression of the sponge and thus facilitating the reception and retention of fluid within its tissues. In other words, an increase in the internal, local work diminishes the amount of visible external work, especially the part performed by an organ in the onward movement of the blood in the circulation. The increase of the inner, chemical work, which is expressed by the severity of the local inflammation, diminishes the amount of general chemical work for the purposes of the organism; that is to say, the greater the degree of inflammation and fever, the greater the impairment of the appetite and power of digestion. Both processes, the chemical and the physical work, exert a reciprocal influence on one another, since the completion of one cycle acts as a stimulus for the beginning of the other.

When, therefore, a sudden increase in the amount of blood necessitates a greater local activity and produces a temporary (relative) inability of the vessel-walls and of the protoplasm in the parenchyma of the lung (the endothelial cells) to effect mechanical removal of the blood, the amount of blood in the suddenly expanded lung cannot, as under normal conditions, be carried back to the circulation through the venous channels. The overfilled vessels must be released by an increase of the local activity and by utilization of all the reserve spaces. The walls of the vessels relax and permit a large mass of material to pass through into the lymph-spaces surrounding the tissue, whereas under ordinary conditions only a small amount of nutritive material sufficient for the nutrition, that is to say, the work of the parts, makes its escape and is therefore readily carried off by the ordinary channels.

The enormously increased activity of the inundated portions of the parenchyma therefore adds to the mass of lymph which accumulates whenever the power of transportation is lost.

That there can be no question of injury to the vessel-wall, in many cases at least, is shown by the fact that after evacuation has been accomplished,—that is, large masses of lymph have been secreted with the production of intra-alveolar and interstitial edema,—complete recovery usually takes place. Hence this increased local secretion of lymph, like excessive salivation after irritation of the chorda tympani, cannot be regarded as a sign of paralysis of the vessel-wall or destruction of the tissue.

The difference between edema in active and in passive hyperemia is simply that in the former case there is a relative insufficiency with a maximum amount of arterial blood, and in the latter, an absolute insufficiency with a minimum amount of arterial blood. In the former case compensation is perfectly performed by the tissue because the lymph-spaces are open, in the latter compensation fails because the venous stasis is already a sign of the great increase in local activity.

Various observers have attributed the occurrence of edema to the employment of aspiration, but this view cannot be correct for all cases, for aspiration under very low pressure and forced evacuation are not the same thing, since the negative pressure can and ought to be regulated and modified to suit the conditions of the particular case.

It is evident that too rapid and copious evacuation of an exudate is dangerous, because it does not permit of a correspondingly rapid expansion of the lung. It can occur only when forced aspiration is employed, and does not follow an ordinary evacuation, which is practically brought about by the normal pressure. But I must insist once more that even when this undesirable event takes place, it is not the result of aspiration itself, but of an incorrect use of the method which, in the hands of an expert and under suitable conditions, is absolutely harmless. Edema of the lung has not rarely been observed even after an ordinary puncture without the use of an aspirating apparatus or with the employment of a small amount of hydrostatic pressure.

[The subject of albuminous expectoration is exhaustively treated in a recent article by Riesman, who completes the literature, which includes several English and American observers, and adds a case of his own.—Ed.]

Too many repetitions of the operation when the indications are not perfectly clear, which are of course followed by rapid reaccumulation of the exudate, produce similar unpleasant results. In debilitated and irritable persons the mere introduction of the trocar, or even the preparatory procedures alone, may lead to asthma and syncopal attacks. In order to escape censure in this respect one should strictly adhere to the rule which I have given above, and immediately stop the flow or even terminate the operation whenever any alarming symptoms of weakness make their appearance or the patient begins to cough violently and persistently.

If the exudate reaccumulates to the same level immediately after evacuation, some time should elapse before puncture is again resorted to. If, on the other hand, the exudate increases slowly and only reaches a moderate degree, evacuation may again be resorted to after a few days, and is then as a rule followed by permanent recovery.

Evacuation of the exudate by means of puncture, in addition to its curative value possesses some importance from a **diagnostic and prognostic** point of view, since the actual condition of the tissue and function of the lungs can be properly ascertained only after the exudate has been removed. A focus in the lung is often discovered which before had escaped detection, either because it was covered up by the exudate or because the lung is able to functionate more perfectly and the auscultatory and percutory phenomena therefore become more distinct. After the evacuation of an exudate the apex not infrequently shows catarrhal phenomena or other important changes in the respiratory murmur which point to the presence of deeper alterations in the tissue.

The evacuation of the exudate is not infrequently followed by an *increase in the amount of sputum*, the examination of which may afford valuable positive or negative information.

In cases of carcinomatosis of the pleura, metastatic tumors have been observed in the wound caused by the cannula (Unverricht).

[Among the possible sequels of thoracocentesis Minciotti mentions the occurrence of pleuritic urticaria, supposedly due to the removal of the upper stratum of the fluid and the subsequent concentration of the micro-organisms and their products in the remaining fluid. He believes that the early appearance of such an eruption indicates the probability of rapid absorption.*—Ed.]

(e) **Incision in Failure of the Exudate to undergo Absorption.**—When the absorbing power of the pleura is lowered and the formation of dense plates appears to be the only obstacle to recovery, I would advise incision whenever hectic fever or any distinct variations of temperature are present.

Even when there is only a suspicion that the pleural disease rests on a tubercular basis, incision is indicated unless there are positive signs of marked involvement of the parenchyma, the more so if we remember the very satisfactory results which follow a mere exploratory incision without any medication in tuberculous peritonitis.

If the complete evacuation of the exudate and the entrance of air into the peritoneal cavity have been followed by good results, even in diseases of tuberculous nature, more may be expected from thoracotomy when the failure to undergo resorption is due merely to the formation of plates. It is certainly the only way to bring about thorough evacuation and gradual separation of the plates and the ultimate expansion of the not yet carnified lung, and it is usually followed by an improvement in the temperature and in the local inflammatory process. If the lung should fail to expand completely after a certain time, an appropriate osteoplastic operation on the thoracic wall will eventually bring about recovery, as in old cases of empyema (see page 961).

We conclude, therefore, that whenever a serous exudate has reaccumulated after repeated tappings and the patient shows loss of strength with abnormal variations in temperature, the cutting operation is to be urgently advised, and we are convinced that many a case can be cured in this way which would otherwise inevitably end in gradual loss of strength or in tuberculosis. [See papers of S. West.—Ed.]

3. TREATMENT OF SIMPLE EMPYEMA.

(a) **Choice of Methods.**—While all the authorities agree that under certain conditions the best method of treating a serous exudate is found in puncture with moderate aspiration, the question whether puncture in the form of permanent aspiration with drainage, after Bülau, is to be preferred in empyema to the radical cutting operation is still the subject of violent controversy.

Although I believe that, according to the old surgical principle "*ubi pus, evacua,*" a collection of pus in the thoracic cavity is always best combated by means of the radical operation, I cannot quite admit the correctness of the observations of certain trustworthy authors who affirm that after simple puncture with irrigation and the employment of Bülau's method of drainage they obtained complete resorption of the exudate, and shall therefore enumerate the various other methods proposed instead of the radical operation.

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* *Gazetta degli Ospedali*, November 10, 1901.

There is, in fact, a great temptation to substitute for operation by incision, which both for the layman and for the physician possesses all the characteristics of a major operation, a more simple procedure, and thus avoid anesthetization and the troublesome after-treatment.

Although the fact that an empyema not infrequently undergoes absorption after simple puncture cannot be adduced as a reason for choosing puncture as the best method of treatment, it cannot be denied that in weak and exhausted individuals, when there is reason to dread the effect of prolonged anesthetization, and the necessary assistance cannot be had, Bülau's method, which certainly fulfils all other therapeutic requirements, possesses many advantages over the cutting operation, especially when the latter is combined with resection of the ribs.

The choice of method should, however, not always be made to depend only on the patient's condition, since patients in a very weak state sometimes bear the cutting operation very well and anesthetization is not absolutely necessary if no resection is done. In any case, if, after the methods by puncture have been tried, the fever does not subside and the symptoms do not show a marked improvement, the radical operation should not be longer delayed, as every additional day diminishes the prospect of proper expansion of the lung and increases the danger of complications.

Hence the most important consideration in deciding on the necessity of operation is "not to let the purulent exudate become too old" (Ewald). And I am inclined to adhere to this principle which presupposes the ability to make an early diagnosis, in spite of the claims of certain statisticians (Runeberg,[13] Holsti[14]) who assert that exudates operated on during the first month require a longer period for their recovery than those which are operated on later.

As the individual conditions no doubt play an important rôle and the ultimate result is determined by the etiology,—whether, for instance, one has to deal with a metapneumonic empyema or with an empyema due to some other cause,—the extent of the disease, and, finally, the circumstance whether it is a primary or secondary affection, the value of the statistics adduced by the above-named authors is difficult to estimate correctly. The facts brought forward in support of their view are not sufficiently convincing to induce me to give up the view that the evacuation of an empyema ought not to be delayed for any length of time.

Hence the importance of determining by an early exploratory puncture the nature of the exudate, its position, etc., and if there are any signs of its being a purulent exudate, such as certain typical variations in temperature, chills, and profuse sweats, not to allow one's self to be deterred from again resorting to the entirely harmless procedure of tapping if the first attempt is followed by a negative result. It often requires three or four punctures and a cannula of greater length and larger caliber to find the right spot, which incidentally shows where to perform the operation (see pages 895 and 946).

As regards the **method of performing the cutting operation** and the **after-treatment,** it is a source of great satisfaction to the author[1] that the views expressed by him in the second edition of Eulenburg's "Real-Encyklopädie" and even earlier [14] in regard to operation and after-treatment of empyema, advocating the *iodoform* treatment and the employment of two large drains to the exclusion of irrigation, have in the main—that is, barring the proposition to limit resection of the ribs to a few cases—been accepted by the profession. Irrigation has now been generally abandoned; only one intercostal space is opened, and the treatment with iodoform, first recommended by the author in the after-treatment of empyema, is becoming more and more general.

[A unique observation is reported by Jacobi.* In a case of pyothorax, after the chest had been opened for the purpose of removing the pus and the pleural cavity washed out, a profuse hemorrhage occurred from numerous bleeding tufts from a few millimeters to one centimeter in diameter. The case, which ended in complete recovery, was neither malignant nor tuberculous. The arteries were uninjured. —Ed.]

Whether a rib is resected or a simple incision made, which latter in the author's experience has always produced the most satisfactory result except in very much neglected cases; whether the incision is made at the deepest part of the pleural space; whether the incision is to be followed by at least one irrigation with an antiseptic solution, depends on circumstances in the individual case and cannot be determined by any general rules.

Very narrow intercostal spaces, multilocular exudates, adhesions, accumulations of pus between the lobes of the lung, abscesses which are often only circumscribed—all these conditions will cause the physician to stop and think, and force him to modify his operation according to the individual case. These points will all be discussed more in detail later on.

Bülau's procedure, as described by Jaffe [12] and Hertz,[12] consists in combining puncture performed under strict antiseptic precautions with permanent aspiration. At the time of puncture a Nélaton catheter is introduced through the cannula, and, after the removal of the cannula, secured in position by means of a cotton and collodion dressing. This is connected with a long rubber tube into which a piece of glass tubing is fitted, and by means of constant siphonage the accumulating secretion is drained into a vessel containing some disinfecting fluid. The low degree of negative pressure (or rather suction) produced in this way offers the most favorable conditions for the gradual expansion of the lung without the accession of air.

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* Association of American Physicians, 1901.

Bülau's method has been received more favorably by clinicians than by surgeons; it has recently been warmly recommended by Leyden and Bohland.[12]

The favorable results obtained by this conservative method of treatment in suitable cases demand its more extensive use in private practice, especially when there is reason to dread the effects of chloroform narcosis, which is desirable although not indispensable, even in mere incision, or when it is desired to avoid a sudden evacuation of the pus, or when, owing to a misconception of the facts, the entrance of air into the thoracic cavity is feared.

One of the chief disadvantages of Bülau's method is the necessity for the patient to maintain the dorsal position for a considerable length of time until the secretion has become much diminished and the cannula or catheter is so firmly fixed in the chest-wall that it will not be dislodged by sudden movements of the body, as in coughing or sneezing. Even if this does happen, it is not difficult to reintroduce the instrument, but the accident is always an unfavorable complication unless the physician lives quite near his patient. The method also has the disadvantage that large shreds of tissue or membranes, which, it is to be remembered, are much less rare than is stated in the text-books, with difficulty pass through the narrow cannula and are apt to give trouble by arresting the flow.

Stoppage of the cannula or of the tube by coagula is best avoided by adopting A. Fraenkel's[13] suggestion of attaching a T-shaped piece of glass tubing to the drainage-tube in the thorax and connecting the free ends with two rubber tubes, one of which acts as a drain while the other is used only when stoppage occurs. In that event the drainage-tube is cut off by a pinch-cock where it leaves the thorax and an antiseptic solution introduced through one of the free ends which, as it flows out at the other, carries with it any coagula that may obstruct it, after which any coagula in the large drainage-tube may be washed out by removing the pinch-cock.

The *method of Bälz and Kashimura*[12] consists in an attempt to further resorption of the fluid by diluting the purulent contents of the pleural cavity with antiseptic solutions until the fluid comes away clear. The exudate is first evacuated by means of a double trocar provided with two rubber tubes, through which the antiseptic solution is then introduced. The procedure is repeated at stated periods until the exudate is absorbed.

Senator[12] has proposed a similar plan. Instead of evacuating all of the purulent fluid, he removes a part of it by means of aspiration and injects a warm antiseptic solution, believing that by diluting the pus it will be more apt to undergo absorption.

To recapitulate once more, we have *three methods of treating empyema*, each of which has its adherents and its advantages, and, as Leyden says, we are fully justified in choosing one of these three methods according to the individual conditions of a particular case, and our own inclination. The most conservative of these three methods is *puncture,* especially as represented by Bülau's siphon-drainage or puncture with irrigation; next comes simple *incision* with the introduction of drainage-tubes which shortly after the operation facilitate evacuation of the pus from the deepest parts and later assist in maintaining a permanent opening in the thoracic wall. The most complicated proceeding is the *radical operation,* which includes resection of one or two ribs and cannot be performed without anesthetization and skilled assistants.

While I am led by my experience to advocate free opening of the pleural cavity by means of an incision, it seems to me that among the methods of puncture Bülau's procedure is the best, as by its employment the fluid is evacuated in accordance with the conditions of pressure within the pleura. It does not force the lung to expand with excessive rapidity, nor does it tend to limit expansion after it has been attained by the introduction of a foreign medium (gaseous or fluid). It does not introduce a new irritant into the pleural cavity, nor does it affect the healing process in any way that does not correspond to individual conditions.

Bülau's procedure imitates the conditions which obtain when an abscess ruptures spontaneously, and is even more effective, since it maintains a permanent opening in the abscess. Nevertheless I would always advise against the use of the method if there are large shreds of membrane which cannot be evacuated, if the patient is distressed by the enforced position, if the pus becomes fetid, or the exudate does not disappear within a reasonable time. Under such conditions the wound should be opened without delay, a thick drainage-tube introduced, and the typical operation by incision, if necessary combined with resection, resorted to.

(**b**) **The Radical Operation.**—There can be no doubt in any physician's mind that the complete cure of an empyema includes not only the removal of the suppuration, but also the approximate restoration of the normal functions of the lung. But as this result depends on a number of factors,—(1) the tonus, the elasticity and distensibility of the organ; (2) the movability of neighboring structures, especially the ribs and muscles of the thorax; and (3) the state of the irritative process within the pleura which determines secretion and absorption,—it is difficult, if not impossible, to estimate directly the comparative values of the various methods of operation, the application of which, according to statistics, varies greatly in individual cases.

In children and young persons, in metapneumonic empyemas, in any case where the presence of a purulent exudate is diagnosed early, any method which affords free outlet to the pus will probably prove successful. Hence a method which yields favorable results in such cases cannot be expected to prove equally successful in elderly persons with partially ossified thorax, in emphysema, in the presence of inflammatory processes in the lung or neighboring organs, or in badly neglected cases.

Unless the material is carefully sifted according to the peculiarities of individual cases, a general statement such as we frequently meet with, that one method which fails to cure after months of treatment is necessarily inferior to another which proves successful after only six weeks, is of very little value. It goes without saying that a method employed after the disease has lasted for some time offers much less chance for recovery than one which is employed immediately after the formation of the empyema, simply because in the former case the lung has lost the power of expanding.

To construct a table of comparative statistics, therefore, only cases of apparently similar character, course, and duration should be utilized, and it must not be forgotten that the most important factors in the statistics are the nutrition and general sanitary conditions of the patient before the development of the disease, and the time when the patient first comes under the care of the physician.

Operation by Simple Incision.—We shall first proceed to describe the simplest proceeding which we have found successful in a series of cases. It is a procedure that is always proper unless the disease has lasted so long as to produce irreparable disturbance in the mechanism of the lung or some marked alterations in the architecture of one-half of the thorax, such as complete obliteration of the intercostal spaces by the approximation or superimposition of the ribs.

The operation should properly be performed under chloroform narcosis, though I have sometimes operated without anesthesia when the patient was very weak or *indicatio vitalis* existed. The anesthesia in the operation by simple incision need not be profound, since the division of the intercostal space and the introduction of drainage-tubes can easily be performed in two or three minutes.

As regards the employment of local anesthesia to avoid chloroform narcosis, I cannot speak from experience. I have no doubt the operation could be performed under local anesthesia, and its application may be very desirable in order to dispense with chloroform narcosis.

Technic.—The incision, unless there is an empyema necessitatis should be made in the **fourth, fifth,** or under special conditions in the **sixth,** intercostal space. The incision should begin at the nipple or anterior axillary line and be carried at least two inches outward.

It is of the greatest importance to make the incision as broad as possible, both to afford a free outlet for the pus and to allow the fingers to be introduced into the thoracic cavity for the purpose of breaking up and removing the false membranes which tend to retard healing. This manipulation, which has recently been again recommended by surgeons (König), is said by many observers greatly to facilitate rapid and complete recovery.

In general, the interference with recovery by these membranes does not appear to me to be very great; I have seen prompt recovery take place in quite a series of cases where there were numerous thick membranous deposits which were not removed mechanically and had not later come away of their own accord. It seems probable, therefore, that they are broken up into minute shreds within the pleural cavity or dissolved by the pus and escape in that form.

Avoiding the larger venous trunks, we divide the skin and first layer of the superficial fibers of the intercostal muscles layer by layer, and cut through the deepest layer of the pleura with a bistoury, whereupon the pus bursts forth in a steady stream. Immediately after introducing the bistoury the wound should be widened, either to the right or to the left, the knife being held in a perpendicular position. The pus is prevented from discharging too rapidly by introducing one or two fingers into the thoracic wound, so as to avoid too sudden alterations of pressure within the thorax.

The deep layers of tissue should be rapidly divided, as this shortens the operation materially; there is no danger whatever of wounding a vessel at this depth.

Leyden has devised a special instrument—the thoracotome—which by means of a spring-blade enables the operator to divide the soft parts at one sweep.

The chief objects to be aimed at in the operation are to afford the freest possible outlet for the pleural secretion and to prevent decomposition. These objects are best attained, as in the case of abscesses, by allowing free play to the natural factors which favor the expansion of the lung and the action of the thoracic muscles, and by avoiding any manipulations within the pleural cavity which only act as irritants and retard the healing process. In most uncomplicated cases all that is required, therefore, is to make a sufficiently large opening to secure complete evacuation of the entire cavity during the first one or two weeks; that is, until the time when complete expansion of the lung is sure to have taken place.

All these indications are sufficiently met by introducing **two drainage-tubes 8 to 10 cm. long,** taking care that the inner extremity does not rest against the thoracic wall, the lung, or the diaphragm. For adults the tube should be about the size of the little finger, the caliber being, of course, varied to suit individual conditions.

We advise the introduction of two drainage-tubes immediately after operation, both because it facilitates the evacuation of the pus and produces a larger opening, and because if one drainage-tube becomes temporarily obstructed, the evacuation can continue through the other one.

The openings in the drainage-tube should be fairly large. As soon as granulations begin to form in the canal, however, no openings need be made in the drainage-tubes, as the granulations would only grow into the openings, and not only occlude the lumen, but also cause hemorrhage whenever the position of the drainage-tube is changed.

To facilitate the introduction of the drainage-tubes when the intercostal spaces are very narrow, the tubes should be greased with vaselin and the inner extremity slightly compressed before insertion. I am in the habit of dipping them in iodoform powder after the vaselin has been put on.

The outer extremity is transfixed by a large safety-pin, through which, or better through the wall of the drainage-tube, a thread is drawn and secured with a knot after being passed around the thorax. To prevent the safety-pin from pressing on the soft parts, a packing of small pieces of cotton smeared with vaselin and dusted with iodoform is interposed.

That the rule to fix the drainage-tube must be observed even in the case of narrow fistulas or an apparently closed fistula is shown by a case which came under my observation, where the pleural cavity appeared to be completely obliterated except for an oblique fistula 10 cm. in length, into which an elastic catheter of the same length could with difficulty be introduced. Although the purulent discharge was so slight that there appeared to be no doubt of the fistula being closed at its inner extremity, a drainage-tube 6 cm. in length, which had not been secured, suddenly disappeared completely within the wound. The fistulous tract had to be laid open for a distance of almost 6 cm. before the lower end of the catheter could be reached and the foreign body removed with a pair of forceps.

If iodoform is used, the **dressing** becomes so simple that any trained nurse can be trusted to attend to it, as there appears to be no necessity of excluding the air. After using *iodoform* for some time I have come to believe that it accomplishes the object sought for in the treatment better than any other medicament. It prevents the secretions of the wound from undergoing decomposition and assists in arresting the discharge without interfering with the complete expansion of the lung. Even a putrid exudate can be rendered odorless with this drug in a very short time.

Since 1879, when I used iodoform for the first time, I have given up irrigation of the pleura. This procedure appears to me to be positively harmful, as it renders asepsis within the cavity an impossibility, favors suppuration and putrefaction, and is apt to lead to intoxication, as is shown, for instance, by the rapid appearance of carbolic acid in the urine after irrigations with carbolic acid; and, lastly, it interferes with early expansion of the lung.

The **dosage** and the intervals between the applications must be regulated by individual indications. In simple uncomplicated cases two to three grams will suffice during the first three or four days; later, smaller amounts at intervals of several days, when the dressing is changed, are sufficient. The powder may be directly dusted into the pleural cavity or it may be introduced by smearing the drainage-tubes with vaselin and dipping them in iodoform so that the powder is carried into the most remote portions of the cavity. After the drainage-tubes have been secured with safety-pins, as already described, they may be dusted with iodoform, after which the dressing, consisting of a thin layer of cotton smeared with vaselin and a thick pad of oakum, is applied, covered with protective, and held in place with a wet bandage or, better, with a gauze bandage. When the dressing is changed, the opening is again dusted with iodoform and a dry dressing reapplied without any other precautions. To prevent the safety-pins from pressing on the wound it is customary to interpose a pad of cotton smeared with vaselin.

I have never observed any symptoms of *intoxication* due to iodoform absorption, and I do not believe that the powder can be absorbed when such a small amount of it is used, providing every opportunity is given the pus to escape from the pleural cavity. In all the observed cases of iodoform poisoning absorption was unquestionably due to the fact that the powder was firmly packed into the wound cavity. Nevertheless, it is always well, even when the drug is used in small doses and with every precaution to prevent mechanical pressure,—that is, by simple dusting,—to be on the alert for the first appearance of the symptoms of iodoform intoxication, which, as is well known, consist of changes in the voice, elevation of temperature, and mental excitement.

It is obvious that both the operation and subsequent treatment have to be modified when the lung tissue is diseased, when other complications arise, or compression has lasted too long.

One should never neglect to keep a careful record of the *temperature* during the period of **after-treatment**. It should be taken three or four times a day, because the smallest degree of obstruction is immediately followed by a change in the general condition, loss of appetite, and a rise in temperature. Even a slight rise in temperature—and when the temperature is generally subnormal a temperature of 37.4° C. (99.3° F.) to 37.5° C. (99.5° F.) is, in my opinion, always a danger-signal unless another cause can be discovered for the rise in temperature—usually indicates retention of the secretion and calls for careful inspection of the dressing and the state of the pleural cavity. In a normal recovery the morning temperature at first should not exceed 36.8° C. (98.2° F.) or 37.0° C. (98.6° F.), the evening temperature being about 37.2° C. (98.9° F.) to 37.3° C. (99.1° F.).

With these precautions, and by changing the dressing at first frequently and later at longer intervals, depending entirely on the rapidity with which the bandages become saturated with pus, rapid expansion of the lung and a marked lessening of the secretion will be achieved. After four or five days, if the healing process is progressing favorably, the dressing will be comparatively free from discharge and need be changed only once in two or three days, the surrounding skin being at the same time anointed with vaselin so as to guard it against eczema, which is often produced by the irritation of the pus. A little later, when the secretion has practically ceased, a light dressing may be applied which will permit the patient to walk about without inconvenience, providing his general condition permits. Recovery is materially aided by a little exercise.

After the drainage-tubes have been removed, the wound may be dressed with *oxid of zinc.* The granulations are rarely so exuberant as to require treatment. If necessary, they may be touched with nitrate of silver, which will destroy any prominences within the fistulous tract and assist the external wound to close.

On the second day after the operation the drainage-tubes, which at first are inserted as far as possible, must be shortened, as all that is necessary is to keep the opening in the thoracic wall patulous and maintain a small canal inside the pleura so as to prevent a portion of the lung near the incision from closing the opening and obstructing the outflow.

A small amount of resistance felt on changing the position of the drainage-tubes does not necessarily mean that the walls of the cavity are beginning to adhere. Often the diaphragm rises with astonishing rapidity, or the lung becomes suddenly inflated, or granules and shreds of membrane make it appear that the cavity has become obliterated, whereas, in reality, it is only the internal opening of the canal in the chest-wall that is occluded.

If the drainage-tubes are changed too soon, and the canal becomes narrowed at one point owing to the premature introduction of very small drainage-tubes, so that the secretion accumulates, the opening can easily be made larger by the introduction of a tent, by means of packing, or with a solid bougie, so that a drainage-tube of any desired size can be introduced.

If the amount of discharge and the complete absence of fever do not sufficiently indicate that closure, which may take place from within outward, has been effected, a small catheter may be inserted through the drainage-tube so as to determine whether it is still patulous. The safest plan is to reduce the length of the drainage-tube to 8 cm. two or three days after the operation, after which it will be gradually extruded by the healing process. When the fistulous canal has been reduced to 4 or 5 cm., the drainage-tube may be replaced by iodoform packing; by this means we can make sure that there are no diverticula remaining to interfere with complete obliteration of the cavity.

An important technical point which deserves a few words of mention is the **position of the incision** under various circumstances. To make two openings is absolutely useless when a single one affords free outlet for the pus and lies at a higher level than the diaphragm. With the exception of a few cases of empyema necessitatis, the operative wound is best made in the **lateral wall.** It should be made in the *posterior surface* of the thorax in the case of an encapsulated exudate that cannot be reached either from in front or from the side. In any case a posterior incision is accompanied by certain disadvantages. It often prevents the patient from lying on his back and the drainage-tubes are more apt to become displaced or occluded.

The incision should never be made lower than the **sixth intercostal space** unless it appears evident that the diaphragm is greatly relaxed and incapable of functionating. If free drainage is secured as deep as the vertebral column, and the accumulating fluid is evacuated by making the patient sit up at frequent intervals, it is not only unnecessary, but distinctly unwise, to make an incision lower than the sixth interspace for the purpose of reaching the dependent portions of the cavity, as the drainage-tube is in constant danger of being bent on itself by the rising of the diaphragm, or of becoming occluded by granulations on the surface of the muscle, especially when short drainage-tubes are used. Both of these accidents, as I have often convinced myself, are apt to be followed by retention of the pus with its necessary consequences: fever, pressure on the expanding lung, depression of the diaphragm, and decomposition of the pleural contents. To avoid these unpleasant consequences it is therefore best to make the incision in the fifth or, at most, in the sixth intercostal space about the middle of the lateral wall, so that the drainage-tube lies loosely on the diaphragm, even when the muscle rises to a considerable height.

Necessity of Resection.—Any elaborate discussion on resection of the ribs will no doubt appear superfluous to many readers, as not only the surgeons, but the vast majority of clinicians who have given their attention to this point, have recently pronounced in favor of the necessity of this operation. Nevertheless the point seems to us to deserve some mention, for the reason that the conditions in private practice are different from what they are in clinics and hospitals.

Not that I wish to take a decided stand against resection of ribs, since the operation is comparatively insignificant if capable assistants are to be had, and it undoubtedly affords a permanent outlet for the pus. I am quite sure that there are cases which absolutely require resection of the ribs; it is a fact, nevertheless, that, although resection was never performed in any of the cases that have come under my observation, even when the intercostal spaces were greatly narrowed, a cure was in every instance effected without the operation.

While the removal of a section of rib is a simple operation for a practised surgeon, and while it effects the evacuation of the pus without any other procedure in every case, providing the lung is able to expand, the operation nevertheless fails to keep the cavity open if closure of the cavity is impossible because compression of the lung has lasted too long. Besides, with the exception of the cases mentioned above, it is always in our power to maintain the opening as large as we please and to keep it patulous as long as may be necessary by the introduction of two drainage-tubes of the necessary length and size; the wound begins to close only when the caliber of the drainage-tubes is purposely changed.

An opening which immediately after operation, on account of the extreme contraction of the intercostal space, barely admits the little finger, and into which even a medium-sized drainage-tube can be introduced only by partially compressing it, may in a few days become so much dilated that even the index-finger can readily pass. Even when granulations are formed in the fistulous canal or in an empyema necessitatis, as they not infrequently are, they may be made to disappear in a short time by the pressure of the drainage-tube, and I am therefore forced to conclude that resection of the ribs, while it cannot always be avoided, is unnecessary and only complicates the operation in the vast majority of cases, if the diagnosis has been correctly made.

Trephining of a rib, which has been highly lauded by Langenbeck, does not appear to possess any advantages over operation by incision or resection, and has never been adopted by the profession.

Irrigation.—In conclusion, a word in regard to irrigation, for which a variety of antiseptic substances—such as carbolic and salicylic acids, boric acid, potassium permanganate and potassium chlorate, bichlorid of mercury, normal salt solution and other aseptic fluids, and, finally, various kinds of astringents—may be utilized.

The advocates of irrigation contend that large shreds of fibrin which interfere with the healing process are removed by irrigation; that irrigation stimulates the layers of the pleura and thus calls forth respiratory movements and cough; that putrefaction can be controlled only by disinfection, and a purulent secretion by astringents.

These statements have all been disproved by some earlier observations of mine, and particularly by a case reported from my clinic by Hertz[13] (see page 956). If the drainage-tube is sufficiently wide, even large masses and shreds of fibrin will be evacuated, and the expansion of the lungs can be accelerated at will by spontaneous respiratory movements and by cough.

It is as difficult to disinfect a cavity with many diverticula as it is to disinfect the inner surface of a putrefying uterus, for the causes of putrid decomposition usually lie deep within the tissue. The possibility of undisturbed evacuation as provided by a free incision with a permanent opening in the thorax, supplemented by the employment of antiseptic substances in powder form, which is most effective in preventing the development of germs, in our own opinion affords the best means of combating decomposition.

According to Aufrecht,[13] irrigations with warm carbolic acid in afebrile cases, and irrigations with warm nitrate of silver solutions (2% to 5%) during the afebrile stage, tend to hasten obliteration of the pleural cavity. I must, however, take exception to this view, as the object of a properly conducted treatment is not to bring about adhesion of the lung, but to effect a cure with the least possible amount of adhesion; and irrigations are more apt to retard than to facilitate the natural process, namely, the approximation of the costal pleura.

If irrigation is insisted upon,—and I repeat that my advice is distinctly against it,—the wound may be kept open, as advised by Fräntzel, after Bardeleben, by a somewhat oval silver cannula guarded at its exterior extremity by a metal plate, like a tracheal cannula. Through this cannula the irrigations may be made in the above-described manner once or twice a day as the necessity of the case requires. By changing the position of the patient and inserting the catheter or drain in various directions, the antiseptic substance can be made to come in contact with the pleural surface. The fluid should never be introduced at random into the cavity, as this would be certain to retard the healing process. It is best not to introduce more than 150 to 200 c.c. at a time, and to evacuate all of this before any more is allowed to flow in. Quite recently Wagner,[13] following the example of Küster,[13] Schede,[13] and others, has recommended the dry method by means of iodoform packing instead of antiseptic irrigation during the after-treatment of a radical operation. The method recommended by König,[13] which consists in raising the patient by his feet in order to facilitate the escape of the irrigating fluid, does not appear to have much in its favor; all such procedures are as useless as the irrigations themselves which they are supposed to reinforce. Great care should always be exercised in using irrigations, because, as the cases I am about to quote tend to show, the fluid is apt to pass through the intact pulmonary pleura or through openings in it and flood the bronchi, and because the process of expansion and healing is greatly assisted by the formation of delicate adhesions, which are, of course, destroyed if the fluid is introduced under pressure.

The injurious effects of irrigation are shown to perfection in the following case [see also reports of cases by Janeway which proved fatal after irrigation with peroxid of hydrogen.—Ed.] which I had the opportunity to observe years ago, though I had no part in the treatment.

Very small abscess. Incision. Irrigation. Putrefaction and complete retraction of the lung with calcification of the thoracic wall.—In the case of an unusually strong workman, whose lungs were unquestionably healthy and who presented the signs of a small pleural exudate, which by exploratory puncture was found to be purulent, thoracotomy was performed and about 50 c.c. of pus evacuated. The cavity was then irrigated with a 3% solution of lukewarm carbolic acid, a custom which then obtained. This was immediately followed by the appearance of carbolic acid in the urine and other signs of mild intoxication, such as may be expected *à priori* if the pleura is healthy. Although there was no obstruction to the outflow of the irrigating fluid from the thorax the lung failed utterly to expand and the secretion presently began to assume a slightly fetid odor.

Through utter failure to recognize the true cause of this result and in accord with the then prevailing views, this delay in the healing process was attributed to insufficient disinfection of the cavity and an attempt was made by increasing the number of irrigations (the patient was washed out three times a day) and using more concentrated solutions to assist the process of healing and arrest the putrefying process. The amount of fluid used in irrigation was also increased and the fluid was allowed to remain for some time in the pleural cavity by hindering its escape after the cavity had been filled as full as possible.

The patient's condition of course became worse and worse, and as the lung still failed to expand, although the antiseptic substances were frequently changed and more concentrated solutions were used, two ribs were resected to afford a freer outlet for the pus, although the thoracic wound was quite large enough. The irrigations were also continued with renewed energy, but in spite of this the patient's condition grew steadily worse. After a further interval of four weeks the cavity was as large as ever and the secretion from the pleura was very abundant and extremely offensive. At the same time the thorax on the affected side began to shrink, and in spite of repeated resections the ribs and intercostal spaces became completely calcified and finally ossified after the patient had suffered for many months, so that at the last operation (resection of the thoracic wall) the pieces had to be chipped out with a chisel.

The patient finally died with the symptoms of hectic fever and amyloid degeneration. At the autopsy the entire half of the thorax was found to be completely ossified, there was complete splenification and carnification of the lung, which was covered by sections of pleura of a centimeter's thickness. The other lung, with the exception of a few small recent bronchopneumonic foci, was quite healthy, so that the unfavorable termination of the disease can only be attributed to the treatment. The persistent irrigations with antiseptic fluid had been unable to prevent decomposition and had rendered expansion of the lung utterly impossible.

Besides this most instructive case, I have observed others in which irrigations were used and which would have followed a similar course if the error had not been recognized in time, the irrigations discontinued, and a more rational form of treatment inaugurated.

A very instructive case of this kind is the following, which is interesting both on account of the encapsulation and formation of loculi, and on account of the fetid odor of the serous exudate.

Multilocular pleurisy after confinement. Putrid, serous, and purulent exudate, associated with abscess of the lung. Diagnosis made after repeated exploratory punctures. Incision. Communication between the pleura and the bronchi. Escape of the irrigating fluid into the trachea with symptoms of asphyxia. Employment of iodoform. Recovery.—A woman, thirty-eight years of age, the mother of eight children, after a difficult labor terminated by manual intervention (twins with placenta prævia), developed symptoms of a right-sided, exudative pleurisy with severe dyspnea, great prostration, and a moderate rise of temperature.

Exploratory puncture in the lateral wall having revealed a serous exudate, while a puncture in the posterior wall of the thorax liberated about 100 c.c. of an extremely fetid purulent fluid, the diagnosis of a putrid pleurisy partially encapsulated by adhesions within the pleura was made. The pleurisy was evidently secondary to an infected embolus in the lung. In spite of unfavorable external conditions, it was decided to perform a radical operation so as to remove the necrotic material and check the further progress of putrefaction.

Although on the next day signs of an extensive dry pleurisy developed in the left side, manifesting itself in marked pleuro-pericardial friction and violent pain, we persevered in our intention and performed the operation by making an incision 5 cm. in length in the lateral wall, corresponding to the fifth intercostal space. After the pleura was divided a small quantity of serous fluid with extremely fetid odor was evacuated, and on palpating with the finger the cavity was found to be filled with a mass of spongy material. The friable inflammatory exudate offered no resistance to the entering finger, and a second cavity was reached which was filled with putrid pus. In this way we succeeded in removing from a large area on the posterior surface of the thorax a mass of degenerated tissue which appeared to be partly composed of necrotic lung tissue. After disinfecting the cavity with carbolic acid and a solution of aluminium acetate, two rubber drainage-tubes, 10 cm. in length and as thick as the little finger, were introduced and the wound dressed with salicylated cotton and oakum. The patient's condition improved immediately after the operation. A little later, however, there was a change for the worse, as in spite of the constant irrigation with all manner of disinfecting fluids the putrefactive process persisted and the discharge was as profuse as ever.

In addition, owing to the destruction of lung tissue, a pulmonary fistula had been formed and the irrigating fluid, even under very low pressure, entered the bronchi and sometimes provoked alarming attacks of dyspnea. At this critical juncture it appeared imperative to discontinue the irrigations and make every effort to check the decomposition and the profuse secretion by introducing some solid substance into the pleural cavity.

As iodoform appeared to be the best substance for this purpose, its use was decided upon, and 5 to 8 grams (about ʒj¼ to ʒij) of the powder were directly thrown into the pleural cavity, or an equal amount was introduced by insufflation through the drainage-tubes by means of an ordinary insufflator. The result was very satisfactory; after a few days the fetid odor disappeared, the discharge ceased, and after two weeks recovery had begun, so that the patient, who showed comparatively good powers of recuperation, was able to leave her bed.

The pleurisy on the other side disappeared within a few days by spontaneous absorption; it was found by exploratory puncture to consist of a moderately large serous exudate without fetid odor.

Convalescence continued without interruption, the iodoform being applied in successively smaller amounts and at longer intervals. Recovery was delayed only by failure to remove the drainage-tubes, which were purposely left in place for some time. As soon as they were removed the fistula rapidly began to close. The entire duration of recovery, from the day of the operation to the complete closure of the fistula, amounted to twelve weeks. This period would probably have been shortened by several weeks if the drainage-tubes had been removed earlier.

The woman's general condition since that time has been in every respect satisfactory, the function of the right lung is entirely normal, and she has given birth to several children without complications.

4. TREATMENT OF THE COMPLICATED FORMS OF EMPYEMA.

(a) **Empyema Necessitatis.**—In operating for pronounced empyema necessitatis the **site of the incision** is a question of the greatest importance. The unanimous opinion of writers appears to be that the thoracotomy must be performed at the site of the empyema necessitatis, and Eichhorst [1] derives the term from this necessity of operating at the spot where the empyema points. But a case operated on by myself and described by A. Moll [13] proves conclusively that the choice of the site for the incision must depend on individual conditions, and that at least one very severe case of empyema necessitatis was to all appearances cured in a very short time by the very fact that the incision was made in another situation.

Extensive purulent pleurisy. Empyema necessitatis below the clavicle.—The patient was a boy sixteen years old, who had had a purulent exudate on the right side for six months. On admission the patient presented a pitiable appearance; he had fever, he was emaciated to a skeleton, the complexion was yellow, the lips were blue, and there was intense edema in the lower extremities and scrotum. The respiration was hurried and sighing; the pulse could scarcely be felt and was about 160. There was great bulging of the right side of the thorax, which remained immovable during respiration. Immediately below the clavicle and extending for about three intercostal spaces on the anterior wall of the thorax was a conspicuous, fluctuating prominence about the size of a man's fist. It was not affected by inspiration, but became more tense during expiration. The intercostal spaces on the right side were enormously dilated, some of them being as wide as two fingers. The cardiac impulse was found between the anterior and posterior axillary lines.

Although the patient was in such a wretched condition that the operation did not appear to offer many chances of recovery, it was nevertheless decided on in the hope of saving his life. On account of the condition of the heart narcosis seemed out of the question, and although there was a distinct empyema necessitatis, it was so peculiarly situated that it offered no chance for the pus to escape. It was, therefore, decided not to open the empyema necessitatis, but to perform thoracotomy in the lower portion of the thorax. The operation was performed in the manner described above by means of an incision 5 cm. in length in the fifth intercostal space and in the anterior axillary line. The opening of the pleural cavity was immediately followed by the escape of large masses of somewhat musty-smelling pus, the evacuation being retarded by alternately tamponing and reopening the wound. Although the above-described prominence diminished somewhat after the greater part of the pus had escaped, it expanded again to the size of a child's head whenever the patient coughed, which he did at short intervals. On the third day, however, the tumor had completely disappeared and auscultation and percussion over the corresponding area showed that the conditions were normal. The patient recovered completely within a very short time (about five weeks).

We have in this case, therefore, complete *restitutio in integrum* at the site of an empyema necessitatis within an incredibly short time, a result which is quite at variance with the prevailing views in regard to the course of such a perforating process in the pleura. As it is generally supposed that an empyema necessitatis is produced by necrosis of the pleura and the various muscular layers, recovery could not be *à priori* expected without opening the infiltrated and purulent area. A further necrosis of the skin over the diseased area, with the formation of a secondary fistula, seemed less probable, and it was for this reason that it was deemed unnecessary to open the abscess at once with a knife.

As, contrary to all expectations, rupture failed to take place and a spontaneous recovery ensued in an astonishingly short time, this case affords an excellent argument for making an incision in some other situation instead of over the site of the empyema necessitatis whenever an opening in that region would, on account of the position of the fistula, diminish the chances for complete evacuation of the pus, which is, of course, the essential factor in re-establishing the expansion of the compressed lung. [If the empyema had been operated upon when the abscess was localized and first pointed on the surface, might it not have been better to have made this the point of election?—Ed.]

The above-described case appears to indicate either that the process of repair in the pleura differs from that in other portions of the body, since even undermined portions of the membrane at once regenerate or become adherent to the pulmonary pleura if a free outlet is provided for the pus at some other spot and the lung is given an opportunity to expand, or else that not every empyema necessitatis is due to necrosis of the pleura.

It is, therefore, more than probable that in cases like the present, characterized by abnormally wide and yielding intercostal spaces, the pressure of the exudate may produce a hernia-like bulging of the soft parts simulating the classic picture of empyema necessitatis; and the great rapidity with which recovery was effected is no doubt to be explained in this way.

(b) **Traumatic and Double Empyema.**—No general rules can be laid down for the treatment of **traumatic empyema** after stab wounds or gunshot wounds, as the indications are determined by the character of the wound, the entrance of foreign bodies or air, injury of bones and neighboring organs or vessels (hemothorax and secondary anemia), as well as the general condition of the subject.

Schede [13] observed putrefaction in three out of eleven cases of stab wounds, and in two out of nine cases of gunshot wounds.

Küster [13] proposes to guard against asepsis in such cases by introducing a long sound into the wound for the purpose of finding the lowest portion of the pleural sac and making a counter-incision at this point so as to permit thorough disinfection of the cavity. Out of twenty cases treated in this way Schede lost only two, one by collapse due to myocarditis and the other by an extravasation of blood into the pericardium.

In my opinion, it is probably better, if the patient is in a weakened condition, to abstain from active interference, or at most to irrigate the cavity, after enlarging the wound so as to remove large pieces of clothing or splinters of bone with the least amount of injury to the parts, and then to apply *iodoform,* which in most cases will suffice to control sepsis. The treatment of all the various complications of traumatic empyema cannot be discussed in this place. The treatment of *hemorrhage* by direct ligation should be attempted only when the patient is in fairly good condition and the chances of finding the blood-vessel appear favorable. As a rule, hemorrhage tends to cease of its own accord, if the lung is not injured, as the latter by expanding prevents the formation of a cavity and favors coagulation of the blood.

In **double empyema,** which is a comparatively rare condition, the operation must be performed on the side of the largest exudate. After the expansion of the lung has been restored it may be necessary to attempt an incision on the other side.

When an empyema is *complicated by a serous effusion on the other side,* the empyema, as the more important disease, should be first attacked by operative measures. Removal of the empyema is, as a rule, followed by absorption of the effusion, as I have repeatedly had occasion to observe. If the serous effusion should fail to show any tendency to undergo absorption, an attempt must of course be made to evacuate it by means of tapping.

(c) Empyema Complicated by Abscess of the Lung.—Owing to the impossibility of deciding whether the pleurisy is the cause of the pulmonary disease, or, as happens much more frequently, some disintegrating process in the lung tissue is the cause of a putrid empyema, I am inclined to adhere to the view expressed many years ago [14] that an exploratory puncture should be performed as early as possible in all doubtful cases of empyema complicated by abscess of the lung, and even to introduce the needle over the site of the pulmonary focus if no signs of a fluid exudate can be discovered.

If by means of puncture it is found that the tissue has undergone purulent or putrid degeneration, I advise thoracotomy even if there is very little fluid, as this method enables us to reach the infected lung tissue directly. Whenever the existence of a putrid or purulent focus in the periphery of the lung is positively established, even though an exploratory puncture of the pleura gives a negative result, operation ought still to be considered, as, owing to the almost constant presence of pleural adhesions over the pulmonary focus, it offers very fair chances of success.

It is often possible to penetrate deep enough to reach the pulmonary focus and thus obtain direct information in regard to the condition of the parenchyma. The introduction of a slender needle into the lung is absolutely harmless, as has been repeatedly shown, providing the necessary aseptic precautions are observed.

The view expressed by me ten years ago, to the effect that in a large series of cases an operation for the cure of a putrid pleurisy has for its principal object the operative treatment of the infectious pulmonary disease which lies at the bottom of the pleurisy, has been completely vindicated, and I am therefore tempted to repeat what I said at that time.

Since a putrid pleurisy in most cases is merely a secondary disease dependent on a mild or severe form of pulmonary infection, the cure of the pleural affection is almost always determined by the course of the morbid process in the lung. There can be no doubt that the morbid process in the pleura—it is produced by direct rupture of the pulmonary focus after necrosis of the pulmonary pleura, or by contiguity, that is, direct spread of the inflammation through the intact pulmonary layer—can be brought to a favorable termination only if the infected focus and the products of the putrefactive inflammatory process are rendered harmless or eliminated through the bronchi or the thoracic wall, and it is clear that the last-mentioned comparatively favorable event—namely, the escape of the infected material through the exterior of the body—is much more apt to ensue if the opening in the pleural cavity is made by the surgeon's knife instead of waiting for spontaneous rupture. Incision enables us to get at the putrid cavity for the purpose of disinfection, and also brings the surface of the diseased lung within the reach of treatment. Once the pleura is opened, remedies such as *iodoform*, for instance, which acts so admirably, can be directly applied to the lung tissue, whereas the ordinary methods of treating disease of the lungs without opening the pleural cavity, by inhalations, etc., offer very doubtful prospects of success, and at best are extremely slow. It is certainly the quickest and most convenient way to bring about complete restitution of the diseased lung tissue and of the pleura, as the necrotic portions are more rapidly removed from the body than they could be by expectoration through the narrow air-passages or through a spontaneous fistula in the thoracic wall.

When it is remembered that in cases of putrid pleurisy secondary to an infectious disease of the lungs the symptoms referable to the exudate are, as a rule, very obscure because the extent of the effusion, as in the case just reported, is apt to be very moderate, while the symptoms of putrefaction and sepsis dominate the picture, one is forced to conclude that the increased facilities for the cure of the infectious pulmonary disease created by operative intervention, much more than the direct treatment of the putrid pleurisy, are instrumental in bringing about a successful termination.

If the correctness of these deductions is conceded, it follows that in all cases of putrid abscess of the lung where the physical signs indicate that the focus is situated in the periphery, thoracotomy should be performed, even though the extent of the pleural exudate is not great enough to necessitate operative intervention, and I would advise operation even in cases in which the physical signs merely show the presence of a superficial focus in the lungs without indicating the existence of a fluid exudate, since the pleura immediately surrounding the diseased focus is always separated from the rest of the membrane by adhesions between the two layers and the chances of success are therefore materially enhanced.

(d) **Treatment of Empyema in Tuberculous Subjects.**—The treatment of serous pleurisy in tuberculous subjects has already been discussed. In regard to the treatment of empyema in phthisical subjects, who not infrequently develop this condition, the opinions of the various authors both as regards the method of treatment and the possibility of operation show considerable divergence, and a discussion of the question is therefore not without value.

Before deciding on a line of treatment under such circumstances the local condition and the general state of the patient must be carefully looked into.

If the development of the exudate has been very rapid and accompanied by comparatively severe symptoms, and if the examination of the lungs before the development of the exudate has failed to show signs of extensive destruction of the tissues, energetic **operative intervention** may offer a very fair prospect of success. A favorable result may be expected if the pus is evacuated by means of an incision and the after-treatment is rational.

Under these circumstances the medical dictum "*remedium anceps melius quam nullum*" ("a double remedy is better than none"), which is so often falsely applied, is quite appropriate. I have seen a number of cases which ended fatally from exhaustion after repeated puncture, in which it was clearly shown at the autopsy that a radical operation performed at the proper time would have offered considerable chances of success, while, as a matter of fact, half-way measures consisting in repeated puncture had been resorted to because an extensive destruction of the lung was erroneously assumed to be present.

The procedure must obviously fail under such conditions, and, each puncture being followed by a more rapid reaccumulation of the exudate, can only result in a rapidly progressing loss of strength. Unless the pulmonary disease is very extensive we would recommend **incision** (see page 933), even in cases of serous exudates, which in spite of repeated puncture constantly tend to reaccumulate and are accompanied by marked variations in temperature.

If a radical operation appears too severe, **Bülau's procedure** should at least be attempted in any doubtful case, as the most important indication in reaccumulations of pus is to provide a permanent outlet.

In cases of **putrid exudate** in individuals suffering from a slight degree of tuberculosis, I would always advise operation, as it is our duty at least to avoid as far as possible the danger of putrefaction.

Against the operative evacuation of serous or purulent exudates in tuberculous subjects the not altogether unfounded objection is sometimes urged that the improved nutrition of the lung which follows the evacuation of the exudate brings about a more rapid development of the tuberculous foci, or even that new foci may be formed by the spread of tubercle bacilli during respiration. It is a fact that evacuation of an exudate has been followed by a more rapid development of destructive processes in the lung, and even by an acute miliary tuberculosis, while, on the other hand, pulmonary disease often becomes temporarily arrested in the presence of a serous or purulent exudate or even of pneumothorax, and this temporary abatement in the symptoms continues even after spontaneous absorption of the exudate or of the air has taken place.

While it cannot be denied, therefore, that at least a temporary improvement in the pulmonary disease may be brought about by the formation of an exudate (I myself have seen striking cases of this kind) it does not justify us in laying down the rule to abandon an operation which is otherwise indicated on account of the mere possibility of this favorable influence on the pulmonary process. The importance of individualizing in the matter of operation in such cases has already been emphasized, and I may point out that the objections referred to might easily be urged against operation in general in all forms of pulmonary disease; for if one is justified in assuming that the extension of the tubercular virus is facilitated by the expansion of the lung which follows evacuation of the exudate, it may with equal justice be assumed that a like operative intervention will be followed by an increase of the virus and micro-organisms causing other inflammatory or infectious processes.

In very advanced cases of tuberculosis it is, of course, advisable to delay operative interference as long as possible, as any operative procedure will be incapable of preventing a fatal termination due to the basal disease. In such cases even puncture should be abandoned and the patient's suffering relieved by liberal doses of morphin, as the latter procedure is, in my experience, quite as effective as puncture performed to save the patient's life. (See Treatment of Pneumothorax.)

XV. MECHANISM OF RE-EXPANSION OF THE LUNG AFTER OPERATION.

Many views have been advanced to explain the mechanism of the re-expansion of the lung after operation; that is, after the formation of an artificial external pneumothorax.

The process of expansion can be studied in its purest form only in experiments, in cases of traumatic pneumothorax, or in empyema of very short duration when the original mechanism is still effective. Since it is clearly shown by experiments that even with a wide thoracic wound the lungs offer considerable resistance to the entrance of air, and are able within a few hours after receipt of the injury to expand even when the air has effected an entrance; since in many patients, both children and adults, a distinct respiratory murmur may be heard over a large portion of the lung on the second day after evacuation of the pus by incision, showing the rapid restoration of the normal function; since, finally, in any favorable case the conditions are practically restored to normal over the entire lung within four to five days, it cannot be assumed that recovery in all cases (or even in the majority of cases observed) is brought about by granulations or even by shrinking of one-half the thorax with contraction of the ribs and displacement of neighboring organs. Since the diaphragm does not become displaced passively by the weight of the exudate, but relaxes as its tonus diminishes in proportion to the stimulus acting on its surface, it does not after evacuation rise in proportion to the amount of weight removed, but rather in proportion to the strength of the returning tonus; that is to say, the rise is an active one, the membrane resumes its regular excursions as far as the periodic variations in the innervations which regulate the tonus permit it to do so.

As the pressure of an exudate of moderate extent does not correspond exactly to its actual weight, so its disappearance does not exert a traction on the diaphragm; both processes merely influence the reflex tonus, and through it the position of the muscle. This explains why, in spite of the removal of the exudate by forced aspiration, the diaphragm fails to resume its former position, and why it does not always become depressed to the extent that would be expected when the effusion is abundant.

Its position depends on the strength of the tonus and the modification of its reflexes produced by the irritation of the exudate. This also explains why the lung is unable to fill out the vacuum created by too rapid aspiration, so that engorgement and extravasation into the tissues result. If the mechanism is in proper working order and a reciprocal increase and decrease of the tonus of all the tissues is made possible, there is no need of adhesions to effect or maintain the expansion of the lung. As the exudate, and with it the stimulus on the pleura which favors or produces the contraction of the lung, diminishes, the lung must expand, even if there is an opening in the chest-wall; for at each expiration it is inflated by the healthy lung, and the inflation does not cease entirely during inspiration, as in the most unfavorable case the amount of air that enters the pleura during the inspiratory distention of the thorax cannot exceed in quantity the amount of expansion of the thoracic cavity. The pressure of the external air is unable, therefore, to compress the lung as long as there is tolerably free inspiratory movement of the intrapulmonary column of air and a sufficient decrease in the tonus.

The inflation of the elastic lung must continue to increase after evacuation of the exudate as long as the resistance is less for the intrapleural medium than for the intrabronchial air, as the latter must overcome the mean expiratory pressure within the trachea, which is greater and is determined by the action of the healthy lung.

It is not true, therefore, that restitution depends either on the formation of adhesions or on the obliteration of the pleura beginning at the hilus and effected by connective tissue, to which Roser [13] ascribes the largest share in the expansion of the lung.

In view of the rapidity with which restitution can take place the last-mentioned possibility cannot be regarded as possessing any great significance. While it is true that inflation of the lung is most pronounced at the hilus, because this region offers the greatest facilities for the entrance of air, complete restoration of the lung to its normal volume unquestionably depends to a large extent on another factor—namely, the gradual relaxation of the maximum tonic contraction of the lung tissue which accompanies the diminution of the exudate.

In the author's opinion, therefore, the expansion of the lung is not due to an internal air pressure. Cessation of the abnormal contraction due to the foreign intrapleural medium enables the air to fill a larger volume of the lung. As at each inspiration the intrapleural pressure diminishes along with the fluid separating the pleural layers, as the relaxation of the lung becomes greater than the dilatation of the thorax and the movements of the lung tissues more rapid than those of the chest-wall, more and more air can, of course, enter the bronchial system and the lung, and a correspondingly greater amount of air will be expelled from the pleural cavity during inspiration.

Incidentally it may be mentioned that as long as the respiratory mechanism remains intact the air in the lung can never become very much rarefied, since normal function presupposes not only the maintenance of the full atmospheric pressure, but also an increased rapidity of the air-current. Any diminution in pressure within the lung, even though it lasted only a minute, would at once exert a reflex influence on the inspiratory movement. Respiration could be carried on only with the greatest difficulty and by the aid of a great reinforcement of the muscular activity. A true diminution of pressure within the respiratory space can occur only when the amount of air that enters is insufficient to maintain a continuous current. If we erroneously speak of a diminution in pressure, it is merely because with our manometric methods too great a resistance is opposed to the current, that is to say, the current is delayed and interrupted so much that there is always a slight lowering of the pressure in the canal.

The positive pressure exerted by the healthy lung during expiration plays an important rôle in the expansion of the lung, as Weissgerber [14] has quite justly observed. This increase in pressure is transmitted to the bronchi of the diseased lung, and after the lung has once been evacuated by an expiratory effort, the mean pressure within the trachea is so great that it cannot be completely evacuated during the next expiration. Thus the expansion of the contracted lung is facilitated in both directions as soon as the normal tone has been re-established and the abnormal distention of the pleural cavity begins to yield.

According to Aufrecht,[13] the healing process after operation in empyema is effected by expansion of the lung during inspiration because the main bronchus is greater in diameter than the drainage-tube between the ribs, the degree of distention being maintained by the formation of adhesions.

This explanation does not apply to all cases. I am in the habit of making a relatively large thoracic wound in all my cases, and of keeping this wound open by means of two drainage-tubes, so that, since the outer opening in the thorax was disproportionately larger than the main bronchus, the lung could not have become so completely and so rapidly expanded as I have always found to be the case.

I believe that, providing the lung is healthy and the removal of a sufficient mass of the exudate at once produces a corresponding relaxation of the tonus by re-establishing a normal innervation, the layers of the pleura become adherent over a relatively large area within a very short time even when the wound in the ribs is quite large. Several factors therefore appear to be at work, and expansion, when once effected, is probably reinforced and permanently maintained only by the formation of very delicate adhesions between the pleura.

A good deal of light is thrown on the manner in which the function of the lung is restored after operation by a case which came under my own observation and has been reported by Herz.[14] It appears to be important enough to deserve a full history.

Extensive putrid exudate. Cardiac degeneration. Rapid expansion of the lung. Death from heart-failure. Autopsy.—A woman, fifty-nine years of age, was admitted in a condition of profound collapse, with marked edema of the extremities, and high fever. Almost the entire left lung was found to be occupied by an exudate which, by exploratory puncture, was shown to be in an advanced stage of putrefaction. To save life the thoracic cavity was at once incised without anesthesia, whereupon several liters of extremely offensive, thick, purulent exudate were evacuated. The usual after-treatment was then begun

On the next day the patient's condition was distinctly improved, the fever had disappeared, the appetite was restored. When the dressing was changed, the discharges were found to be almost odorless, and on the fourth day the respiratory movements in the entire left lung were quite active. Although the local conditions improved so fast that seven days after the operation the discharges were reduced to a minimum, the offensive odor disappeared entirely, and one drain could be dispensed with, while the other was materially shortened, the symptoms of cardiac weakness still persisted. The edema became more marked and the patient died eight days after the operation in sudden collapse preceded by several mild attacks of pulmonary edema.

At the autopsy the operative wound was enlarged and the patient's fifth rib resected. This exposed a cavity only 3 c.c. in volume which represented the remains of the entire original empyema cavity, and contained a trace of absolutely odorless secretion. The cavity was bounded by the diaphragm, the re-expanded lung, only the lowest portion of which was atelectatic, and a thick plate separating the empyema cavity from the pericardium which was somewhat displaced to the left. The entire left lung, with the exception of the lowest portion, was adherent to the costal pleura by a number of flat adhesions no thicker than cobwebs; a few adhesions were also found in the right lung, which was somewhat emphysematous. Both lungs were very edematous and the bronchi showed marked catarrhal changes. The heart before being opened appeared very flabby and somewhat enlarged. The ventricles, especially the right, were greatly dilated, and their walls extremely thin. The muscle was friable, of a brownish-yellow color, and showed advanced fatty degeneration. No cause was found for the primary putrefaction of the exudate; there was no putrid bronchitis, no embolism or gangrenous foci.

Discussion: It appears by the autopsy that in a patient with advanced cardiac insufficiency suffering from putrid empyema, where operation was performed only to save life and without hope of permanent success, complete re-expansion of the lung occurred within a very few days, the layers of the pleura being united in their entire circumference by extremely delicate (breath-like) adhesions.

This case shows not only the brilliant results to be obtained in putrid empyema by simple incision, but also gives a distinct clue to the mechanism of recovery. It shows that the healing process is not brought about by granulation and the formation of thick plates, as is usually supposed, and proves that, providing only the lung is capable of expanding, expansion when once begun is maintained and reinforced by the formation of extremely delicate adhesions.

As the only adhesions were found over the pleural surface of the heart, where they might have been due to the irritation of the drainage-tubes or possibly to the action of an especially severe local inflammation, we may conclude that the formation of abundant granulations, which is generally regarded as the most important factor in the process of repair, is not only not necessary, but actually disturbs the normal healing process. We therefore agree with Herz[14] that the only *sine qua non* for recovery after maintenance of a free outlet for the pus is the power of the lung to expand.

XVI. POST-OPERATIVE COURSE AND COMPLICATIONS.

A marked improvement has taken place of late years in the statistics of operation for empyema, the percentage of recoveries being between 84% and 90%. The value of these figures is, however, impaired by the fact that all the cases reported by individual observers cannot be directly compared. The questions in dispute will, it is hoped, be materially elucidated by the investigation set in motion by Leyden. The length of time required for recovery after operation cannot be expressed in numbers, as it is modified in each case by a variety of factors. I have seen complete recovery in children within three weeks, while in other cases two to four months were required. The earlier the operation, the shorter will be the recovery, although complete closure of the wound is often delayed for some time, as the outer extremity of the fistulous canal often shows a certain torpidity and continues for a long time to secrete a few drops of pus.

If the lung is healthy and the operation performed early, complete recovery without deformity of the thorax may result, providing the cases receive the proper after-treatment.

In young persons, when the exudate has been evacuated early, there will be during the first months after the operation a certain degree of interference with the respiration and a slight dulness in the lower and lateral portions, but no distortion of the vertebral column; in older persons and when operation is performed late, a certain *deformity of the thorax* with a diminution of the respiratory excursions is to be observed for some time after recovery. In such cases there is also, as a rule, a *scoliosis* of the vertebral column toward the diseased side and the scapula on the corresponding half of the body is depressed.

If irrigations are used, or complications develop, a permanent *pleural fistula* may result. This condition of the formation of a purulent recess within the pleura is inevitable if the lung has lost the power of expanding.

One of the most unfavorable complications which occur when irrigation with disinfecting solution is employed is *intoxication* by absorption of the antiseptic material. The healthy pleura, as is well known, possesses considerable powers of absorption, and even an inflamed pleura, although it may be unable to absorb the albuminous fluid of the exudate, is quite capable of taking up large amounts of various materials which are introduced into the pleural cavity, or of excreting them from the blood into the exudate.

Such substances may also be absorbed by the lung if a communication exists between the lung and the pleural cavity. Whether in such cases there is always a pulmonary fistula, or whether absorption can be effected by an intact pleura pulmonaris, is difficult to decide. In one case at least (reported on page 947) the fluid entered the bronchi directly and produced alarming symptoms of asphyxia. The patients often state that they suddenly taste the drug in the mouth, a phenomenon that may be due either to the passage of the fluid from the bronchi into the pharynx and the distribution of the nerves of taste, or to the fact that certain substances taken up by the pleura enter the blood-current and are eliminated in the saliva. The latter supposition, to judge from numerous observations, appears to be the more probable one.

Carbolic and salicylic acids have frequently been observed in the urine; Lépine once saw a nephritis following irrigation with chlorid of zinc.

Various other grave accidents and cases of *sudden death* (see page 880) used to be observed much more frequently when irrigation of the pleura was more regularly employed than it is at present, probably because the patients were exhausted by the persistent suppuration directly attributable to the irrigations and by the constantly recurring irritation of the pleura and vagus endings, kept up by the introduction of fluids when the strength of the dose was not always regulated (pleural reflexes). I myself observed a fatal case of this kind where no satisfactory explanation for the cause of death was found at the autopsy, unless it were a marked anemia of all the organs and a certain degree of fatty degeneration of the cardiac muscle.

The patient showed all the signs of extreme anemia and marked loss of strength. He had been treated by irrigations for many months without producing any noticeable dilatation of the lung or even diminution of the discharge, which was purulent and very profuse. A weak lukewarm solution of permanganate of potassium was used one day while he was sitting up, and before a quarter of a liter of fluid had entered the cavity he was seized with syncope and convulsions, and died a few minutes later with all the signs of acute heart failure.

Leichtenstern[8] has made a careful analysis of the cases of sudden death in pleural exudate, based on an extensive study of the literature (see page 881), and v. Dusch has expressed the opinion that pieces of thrombi become separated from the compressed lung by the irrigations and enter the circulation, where they produce embolism of the larger arteries.

This cause, in our opinion, can only be responsible for very few cases; in most, death is brought about by a sudden disturbance of the circulation, by anemia of the heart or brain, by reflex inhibition of the vagus or of the respiratory center, or by sudden heart failure.

Cases of sudden death after incision usually occur in patients who are operated upon as a last resort, and the possibility of death occurring even without operation must therefore be taken into account. Death in such cases may also be due to shock, especially when it occurs in patients who are exhausted by protracted disease.

Convulsive attacks accompanying the irrigations of the pleura have been described by Auberue[7]; they are sometimes followed by syncope and death; in other cases the patients recover after a short interval without any bad consequences. The convulsive movements are said to be more pronounced on the side corresponding to the diseased pleura. Milder cases in which the convulsions are restricted to one-half the body, usually the affected side, are described by Weill[7] as *hemichorée pleurétique.*

In some cases permanent paralysis of the affected side of the body and, in rare cases, anesthesia of the parts result; the arm is usually affected more than the leg. Lépine observed paralysis of the arm of the affected side, with muscular atrophy.

Among the *accidents* which may occur during irrigation or in cases where catheters or drainage-tubes are introduced into the thorax after an empyema operation, must be mentioned the entrance of some object into the pleural cavity, such as instruments, sponges, or dressing material, and it is therefore important to see that all instruments and dressings are properly secured. Rubber drainage-tubes and catheters should never be introduced into the wound, even when the opening is small and the canal is short, without passing a double thread through the outer extremity and fastening it to the chest-wall with strips of adhesive plaster, or, what is even better, tying them around the entire thorax. To secure the drainage-tube by means of a stitch passed through the skin, as is the practice of many operators, seems to me an unnecessary cruelty.

An interesting case of death from the entrance of a foreign body into the thoracic cavity during an empyema operation is found in the Memoires of Madame du Hausset,* the lady-in-waiting of Madame de Pompadour.

The young Chevalier de Montaigu, who was a great favorite with the Dauphin and had been brought up with him, developed an empyema which necessitated thoracotomy. Although the operation was successful, the patient's condition soon afterward became much worse, and he died with all the appearances of extreme dyspnea, although no cause for this unfortunate termination of an apparently successful operation could be found. Owing to the peculiar nature of the case and the interest of the Dauphin, the body was opened and there was found in the chest a piece of the leaden syringe with which, according to the custom of that time, certain decoctions had been introduced into the thoracic cavity.

"The surgeon had not acknowledged his negligence and the patient became its victim." With these laconic words, which contain a wholesome admonition to future generations of surgeons and emphasize the dangers of irrigation, the authoress closes her account of the affair.

Many authors mention rapid *putrefaction of the cavity* among the consequences of operation. Cases are also said to occur where, in spite of all possible precautions, it is found impossible to stop the advance of the putrefactive process within the pleura. If such cases have never come within my observation, it is, I believe, because of my custom of operating as soon as the presence of pus has been determined by exploratory puncture, without injuring the tissues by irrigation.

If the healing process is delayed merely by the formation of a *fistula* with callous edges, the fistula may be laid open or scraped with a curet. Since, however, these fistulas often become callous simply because of a small cavity at their central extremity which can only be closed by granulation, and is prevented from closing by the pressure of the pus when it is retained within the fistula by an obstruction at the external extremity, it is always advisable, after dilating the external fistula in its entire course, to introduce a catheter down to the bottom before resorting to more radical measures. In this way a free outlet for the pus is secured, the dilatation of the small central diverticulum or cavity by the accumulated pus is arrested, and the opening is slowly closed by granulations which ultimately force the catheter outward.

I once effected a cure in a case in which there was a fistula 17 cm. in length of three years' standing, traversing the entire pleural cavity, which in spite of dilatation with bougies and the introduction of short drainage-tubes persisted in closing and preventing the discharge of the secretions. By introducing a drainage-tube as long as the entire canal and allowing it to be forced out by the granulations forming at the central extremity, the fistula was closed within six weeks. In this case there was evidently a large diverticulum at the inner extremity of the canal, and the retention of pus had to persist as long as the peripheral portion of the track kept closing up by granulations forming behind it; that is to say, to the central side of the drainage-tube, which was too short. As soon as the canal was kept open by the introduction of a drainage-tube of sufficient length to reach to the end, a free outlet was provided for the pus contained in the cavity, the cavity diminished and finally became closed, after which the fistulous tract also could become obliterated. The case seems to us important because of the striking proof which it affords of the correctness of our views. Although thoracotomy with resection of the ribs had been performed, the neglect of certain important principles during the after-treatment was punished by the formation of a permanent fistula of considerable length which was ultimately closed without operative interference.

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* "Mémoires de Mme. du Hausset," publiés par Hippolyte Fournier, Paris, 1891.

Whenever from one cause or another a *permanent cavity* is formed (the chief cause, if there is a free outlet for the pus, is insufficient expansion of the lung), the size of the cavity should be carefully determined by introducing a fluid through a two-way catheter after closing one of its openings. If during a period of about two weeks the cavity is found to contain the same quantity of fluid, though it does not exceed two or three tablespoonfuls, resection of one or more ribs corresponding to the outer surface of the cavity should at once be resorted to. The injection of such substances as potassium iodid or tincture of iodin, which have been much lauded as stimulants to the formation of granulations, though in reality they possess no such virtue, is worse than useless. The only remedy in such cases is resection of the chest-wall, and the operation is both easy and promising if no calcareous deposits have formed between the ribs or, as even happens sometimes, the soft parts have not undergone ossification. Resection ought to be performed near the mouth of the fistula.

I do not consider it advisable to make a counter-opening, as was formerly done, to facilitate the escape of the pus. Resection should be quite free, so that the opening may be closed by the soft parts coming into immediate contact with the surface of the lung.

Resection should not, as is generally believed, merely fulfil the purpose of allowing the pus to escape, as this can be equally well accomplished by means of an open fistulous tract. The operation should include removal of that part of the chest-wall which, owing to its concavity, is unable to adapt itself to the surface of the retracted lung.

The greater the cavity, the larger the section of chest-wall to be removed. This should be an invariable rule, as it is the only way to gain the desired end without injuring the patient by profuse suppuration or repeated operations. In addition to securing a free outlet for the pus, the complete removal of the cavity should be aimed at.

On this principle the so-called **Estlander's operation,** consisting in the resection of large portions of the chest-wall, has recently been materially elaborated, especially by Schede.[13] Schede's operation fulfils a hitherto unattainable requirement—namely, to bring about closure of the thoracic wall when the lung has lost its power of expanding, and the rigid walls of the pleural cavity are unable to adapt themselves to that organ. By removing not only the ribs, but also the rigid intercostal spaces over the entire extent of the empyema cavity, a flap consisting of skin, muscle, and scapula is obtained which readily adapts itself to the lung and becomes adherent to it. Recovery by this method is so perfect that after cicatrization is completed and the patient can be allowed to use his arms, the lung gradually expands and respiration is fairly well re-established. The mutilated thorax gradually regains its normal aspect and the scoliosis of the vertebral column disappears.

In this way Schede [13] succeeded in curing five out of seven patients —an unexpectedly good result when it is considered that these patients would otherwise have fallen victims to amyloid degeneration.

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B. PNEUMOTHORAX.

I. DEFINITION AND HISTORICAL NOTE.

The accumulation of atmospheric air or gas in the pleural cavity is designated pneumothorax, while the combination of fluid and air is expressed by a corresponding additional prefix, the temporal or causal relationship of the phenomena being at the same time indicated by the relative position of the words. Thus, we speak of *pyopneumothorax* and hematopneumothorax or *hemopneumothorax* when the condition was preceded by the presence of a purulent or bloody exudate, while the terms *pneumopyothorax* and *pneumohemothorax* indicate a primary escape of air which was the cause of the accumulation of pus or blood.

The terms pneumoserothorax and pneumohydrothorax have been proposed by Senator and Weil for serofibrinous or simple serous exudates caused by the escape of air.

This subtle distinction can, however, be carried out only in cases where the progress of the phenomena is under observation from the very beginning. In all other cases it is difficult without an exploratory puncture to arrive at any conclusion in regard to the temporal relation between the morbid processes, as the symptoms produced by a small amount of gas in the presence of larger masses of fluid are very slight, and, conversely, the presence of large amounts of gas makes it difficult, if not impossible, to demonstrate the presence of smaller masses of fluid.

It may not be out of place to remark that in no other clinical condition has the zeal for classification and the passion for physical diagnosis led to such hair-splitting distinctions as in pneumothorax. A modern clinician who by means of exploratory puncture or by evacuating an exudate can at once secure a clear understanding of the conditions, and the physician who regards diagnosis not as an object in itself but as the foundation for prognosis and treatment, find it difficult to realize a period which, proud of its achievements in physical diagnosis, was constantly striving to discover additional shades of difference in the auscultatory signs and to treasure up rare conditions, which might interest the students of pathologic anatomy, but could be of no practical value to the physician.

That in very ancient times the accumulation of air and fluid within the pleural sac was known and diagnosed is indicated by the so-called *succussion splash*, with which the name of Hippocrates is still linked (*succussio Hippocratis*), and we have observations by Morgagni and others to prove that the sign did not fall into oblivion during later ages.

The term pneumothorax was originally used by Itard, and the doctrine of pneumothorax was elaborated by Laennec to such a degree of perfection that only a few immaterial details remained for the contribution of future authors.

In spite of many painstaking efforts to investigate the mechanism and cause of an accumulation of air within the pleural cavity, many an enigma is yet to be explained owing to the difficulty of tracing the biologic processes to their physical basis. Even in the domain of physical diagnosis, where many obscurities have been cleared up by the pioneer work of Skoda, many puzzling questions still remain to be solved. Physical diagnosis indeed is far from deserving its name.

II. ETIOLOGY AND PATHOGENESIS.

Cases of pneumothorax may be divided into **three groups,** the two first of which may be designated accumulation of air through fistula formation, and the last as closed pneumothorax—in part also pneumothorax from internal gas production.

The cases belonging to the first class, which may also be termed **pneumothorax with an external fistula,** are produced by perforating wounds of the outer chest-wall, and are in a sense typified in the operation for empyema. Spontaneous evacuation of pus from the pleural cavity or empyema necessitatis with fistula formation is only very rarely followed by the formation of a pneumothorax, because the opening is so narrow that the discharge of pus is usually proportional to the pressure within the pleura. The expansion of the lung corresponds only to the diminution of the excess of pressure, so that there is no possibility of the air being drawn in from without to fill the cavity produced between the layers of the pleura; but with a large opening like an operative wound the expansion of the lung cannot keep pace with the rapid evacuation of the pus, and the evacuation of the pleural cavity can only be brought about by the entrance of air from without.

In the second form, **pneumothorax with an internal fistula,** the air finds access through perforations in the pulmonary pleura and parenchyma of the lung, or through a communication with the air-containing organs of the mediastinum or of the peritoneal cavity.

If the fistula becomes closed, an example of the third class or **closed pneumothorax** is produced; this may, however, also be formed without any demonstrable external or internal fistula. In such a case the air is either derived from the decomposition of the exudate, or it enters the pleura from the torn subserous connective tissue, as in cutaneous emphysema, or it is possible that a secretion of gas by the lymph-spaces of the pulmonary pleura takes place in some as yet unknown manner.

In a great many cases it is, of course, difficult to determine the true pathogenesis of a closed pneumothorax, as perforations and lacerations in the alveoli of the lung may heal over completely in a short time and altogether escape detection. Even in the costal pleura a laceration the result of an emphysema of the subserous tissue may be subsequently very difficult to find.

That cases of spontaneous gas production do in fact occur is shown by observations reported in the literature, as well as by some of my own. In these cases a sudden accumulation of air manifested itself very clearly at first in a perfectly healthy lung, and either became arrested or rapidly underwent absorption. In the complete absence of any signs of pulmonary disease both before and after the development of the condition, it must be assumed that a pulmono-pleural fistula which has in some unexplainable way been formed became closed in some equally unexplainable manner, or that the gas escaped without any solution of continuity. So far we are without positive proof that a pneumothorax may be produced by this species of gas secretion, and the complicated observations which bear on this point must be received with caution (see pages 975 *et seq.*).

The following is an instructive case of probable traumatic internal pneumothorax in a perfectly healthy lung ending in rapid and complete recovery:

Internal pneumothorax without demonstrable fistula. Distinct metallic phenomena. Recovery without exudate.—X., aged fifteen, school-boy, belongs to a perfectly healthy family and has always been healthy himself. After severe exertion on the parallel bars he suddenly felt violent stabbing pains over the center of the chest and over the left side, with increasing dyspnea on movement. The patient at once went to bed, and when I saw him the next day he showed, when in the recumbent position, no signs of dyspnea in the alæ of the nose or muscles of the neck; there was no cyanosis and no reduction in the temperature of the extremities. The appetite was good, the pulse was not accelerated, the respiratory rate varied between 20 and 24 per minute.

The left half of the thorax appeared to be somewhat dilated, although there was no bulging of the intercostal spaces. The respiratory excursions on this side were materially diminished, the apex-beat was only faintly visible, and nothing abnormal was to be seen in the abdomen. There was certainly no dislocation of the liver.

On percussion there was heard on the left side, as far down as the third intercostal space, both anteriorly and posteriorly, a very loud and deep non-tympanitic note compared to which that of the other half of the thorax appeared dull. Below the fourth rib there was no marked deviation from the normal. Traube's semilunar space was not diminished and there was no dulness over the postero-inferior portions of the lung.

On auscultation of the left lung no abnormalities beyond a slight accentuation of the vesicular murmur were obtained. Over the entire upper portion of the left lung the respiratory murmur was inaudible during ordinary breathing. When the patient was asked to breathe deeply, distinct amphoric breathing with a high metallic adventitious sound was heard. Beautiful metallic phenomena were also obtained when the patient coughed, and from time to time a very distinct *tintement métallique* was heard. No succussion splash was elicited by shaking the patient; on the other hand, a beautiful metallic echo (after-sound) was elicited over the entire anterior surface of the thorax by striking the pleximeter with the handle of the plessor. From the fourth intercostal space down, both posteriorly and on the lateral wall, there was weak vesicular breathing, gradually changing into the above-described amphoric type. The heart sounds were accompanied by a very slight metallic overtone (*Beiklang*). Under simple symptomatic treatment all these phenomena disappeared within a few days.

On the fourth day the patient's condition was entirely normal and he was able to leave his bed, and since that time, for a period of ten years, has never been attacked by any pulmonary disease.

In view of the undoubted occurrence of gas production in exudates in all cavities of the body there does not appear to be, in my opinion, any valid objection to the view maintained by Biermer and Senator, that gas may be produced by the entrance of germs (bacteria) into a pleural cavity,—which, so far as our methods are concerned, must be regarded as closed,—without any atmospheric air necessarily gaining access to the cavity.

The question why this air does not at once become reabsorbed is no answer to the argument, for neither the air nor the exudate from which it is derived can be absorbed as long as the inflammatory condition of the serous membrane persists, and the very tendency to secrete an exudate and allow it to accumulate in the available space presupposes a marked reduction in the power of absorption.

Since, therefore, the resorptive processes are completely abolished during the period of exudation, especially as far as the complicated albuminous bodies are concerned, a double opportunity is afforded for the entrance and colonization of micro-organisms; for the blood-supply, on the one hand, is greatly increased, and, on the other hand, there is a greater obstacle to the removal of the deleterious substances —reason enough for the free action of gas-producing micro-organisms on the pleural contents.

In addition to this, the spaces and clefts within the tissues of the irritated pleura are widely patulous and offer little resistance to the passage of foreign elements, all of which considerations abundantly justify us in entertaining the possibility of gas production without solution of continuity or communication with the external atmosphere.

Nor does it seem to us impossible that, even without the entrance of micro-organisms, gas may be spontaneously developed from the fluid, either by the absorbed gas being liberated under certain conditions of pressure and temperature, or by carbonic acid and other forms of gas becoming liberated in the form of salts containing carbonic acid, or in some other way by chemical decomposition. When the vital influence of the tissues on their environment and their contents becomes impaired by the energy developed in the performance of the essential, local (interstitial) tissue work, when the cohesion of the parts is relaxed, some special form of decomposition or rearrangement of molecules may be assumed to take place in the fluid contents, which eventuates in the production of gas. Even in the arteries free gas occasionally develops after death and, under certain circumstances, even during life.

I agree with Senator that the possibility suggested by Bucquoy of gas being produced in an exudate after the pressure has been lowered by the removal of a few syringefuls of fluid, does not deserve much consideration, if only for the reason that diminution of the exudate is not always identical with diminution of the pressure on its surface, by means of which the excess of pressure in the pleural cavity is said to be transmitted to the internal gas pressure—namely, that of the gases in solution. Such a result would presuppose absolute rigidity of the chest-wall, inability of the lung to expand, the impossibility of neighboring organs being displaced, and a marked diminution of the pleural contents, a series of factors which are probably never present at one time during life, or, if they are present, give rise to some other conditions favoring the entrance or formation of gas. After death, when the tonus is entirely abolished, this formation of gas from the fluids of the body is a constant occurrence. It is all but certain, in fact, that the postmortem emptying of the arteries is chiefly brought about by the pressure of the gas generated from the blood (see page 916).

Hence in the not infrequent cases where no perforation is to be found, it may be assumed that gas is spontaneously generated by a rearrangement or change in combination of the component parts of the fluid, or by the action of the germs of putrefaction, since the composition of the gas, as a rule, does not indicate any similarity with atmospheric air.

According to Ewald's investigations, the partial pressure of the carbonic acid, which is the most common gas found in pleuritic fluids, is very slight. In serous exudates it varies between 7.5% and 11.5%, and does not exceed 20% in purulent exudates. This point will be referred to again further on.

Among the causes favoring **entrance of air from within** must be especially mentioned *tuberculous disease* of the lung. Communications between the air-passages and the pleura are produced by the disintegrating process whenever such a result is not prevented by adhesions between the pleural layers.

Many authors believe that such a rupture of degenerated lung tissue or of a large cavity is always produced by a paroxysm of coughing or some other sudden movement, such as sneezing, bearing-down, etc., which directly act on the parenchyma of the lung. I cannot agree with this view, for I have seen pneumothorax develop in a large number of cases quite slowly and gradually and without any violent phenomena, and cases are known where rupture occurred not in the region of a large cavity, but in a small cheesy focus surrounding a bronchus.

I have often observed the entire absence of pleural adhesions over very large cavities, which presented the peculiar jelly-like movement of the surface as soon as the lung was exposed.

Unless dense adhesions are found over the entire pleura when the organ is removed from the body, the escape of air cannot be attributed to the bursting of a cavity, for a weak spot in the organ is obviously much more apt to be torn during the removal of the lung postmortem than during life. If death occurs several hours or days after the entrance of air, the rupture of a cavity or of a pulmonary focus can, in our opinion, be regarded as the cause of the pneumothorax only when numerous fresh fibrin deposits are found immediately surrounding the opening.

In addition to the caseous process, other forms of the pulmonary disease, *abscess* and, particularly, *gangrene* of the lung, may lead to perforation of the necrotic pleura. Gangrenous areas produced by infectious emboli or pneumonia are particularly apt to be found on the surface, or, if they are central, always extend as far as the periphery.

In these cases perforation is usually preceded by the formation of serous or purulent, and almost always offensive exudate, just as in peritonitis, where the formation of gas is, as a rule, preceded by a putrid suppuration of some standing.

Pneumothorax following putrid pleurisy is a comparatively rare occurrence, because the layers of the pleura early become adherent and prevent rupture. I have, however, observed this complication once or twice, and it is to be remembered that a perforation is not always required for the production of gas, which may be generated in purulent or serous fluids by the action of gas-producing micro-organisms or some other cause. (See May and Gebhard's case.)

As pneumothorax is rarely produced by the formation of a spontaneous external pulmonary fistula (empyema necessitatis), where the escape of the pus is in proportion to the rise of the pressure and is not accompanied by the entrance of air, so it is equally rare after the formation of an **internal pulmonary fistula** by perforation of an exudate into the bronchi, probably because the size of the opening and the pressure conditions are such as to render the simultaneous escape of pus and entrance of air impossible. To explain this, there is no need of assuming that there is a special form of fistula, a so-called valvular closure, any more than to explain the fact that it is impossible to drink out of a bottle with a very small opening or that water cannot flow through a very small tube unless a second opening is made to allow the air to enter.

That an escape of air may take place through the spontaneous laceration of an alveolus (in emphysema or even in an apparently healthy lung) by a sudden insult to the thoracic organs, and also under certain as yet unknown local conditions, has already been mentioned. Extravasations of blood or hemorrhages within the lung tissue may also be the cause of a slight tissue necrosis ending in the formation of a pleuro-pulmonary fistula.

Traube asserts that he has observed pneumothorax after severe concussion of the entire body without any demonstrable injury to the ribs.

That the escape of air necessarily demands a distinct laceration in the lung appears to me very doubtful, notwithstanding such a basis is by present opinion regarded as indispensable. It appears to be proved by experimental investigations concerning the pleura, and particularly by certain processes in the intestinal tract, that membranes are capable of secreting gas or that gas is eliminated from the vessels through clefts and open spaces, and even through the cells themselves, so that it cannot be absolutely denied that air may under abnormal conditions be generated by the pleura by a kind of secretory process. Although the question has not as yet been settled, it seems to us not improbable that in many cases where the most careful examination failed to demonstrate any injury of the pulmonary or costal pleura the escape of air is to be attributed to some temporary impairment, irritation, or paralysis of the pleura affecting its power of secreting gas or preventing the escape of gas. The question is important enough to merit further experimental investigation.

Disregarding certain mechanical conditions which prevent a lowering of the pressure at the perforation, the escape of gas is dependent upon the character of the opening and the conditions of the pulmonary tissue, the pressure changes incident to respiration, the character of the exudate, and possibly also the height of the column of fluid. The thicker and more tenacious the pleural fluid, the denser and more extensive are the adhesions and the greater the resistance offered to the escape of gas. Thus, as Traube has observed, an extensive exudate may be expectorated through the spongy lung tissue without any air entering the pleural cavity. In such a case the failure of pneumothorax to develop can only be explained by assuming that, owing to the rapid inflation and perfect elasticity of the non-compressed portions and the atelectasis of the compressed portions through which the exudate passes, the pressure at the site of perforation or the local obstacle to the air-current is always much greater even during inspiration than it is in other portions of the lung.

Pneumothorax would probably be produced more often were it not that owing to the very free communication of the bronchial systems of both lungs the differences during inspiration and expiration are usually neutralized at some point distant from the site of perforation. If the diseased lung fails to respond to the traction of the inspiratory muscles, more air is forced into the healthy lung during inspiration, while during expiration the expelled air encounters less resistance in the large air-passages than in the diseased or collapsed inflammatory portions of the lung. For this reason pneumothorax is not produced in many cases in which its occurrence would be expected on theoretic grounds; or if it is produced, its volume diminishes more rapidly than ought to be expected.

No accurate data can be given in regard to the effect of the various tissue changes on the production of pneumothorax, as we depend altogether on the statistics of hospitals, where, of course, chronic pulmonary disease is far more common than any other etiologic factor. It may, however, be asserted with tolerable accuracy that the number of cases due to tuberculosis is from ten to fifteen times as great as that of all other forms, among which gangrene of the lung again predominates (Morse).

According to accurate, although not very extensive, statistics by Weil, from 8% to 10% of phthisical subjects show symptoms of pneumothorax, the two sexes being equally represented.

The question whether **acute** or **chronic** cases of phthisis show a greater disposition to pneumothorax has also been investigated. No definite conclusion can, however, in our opinion, be arrived at, because it is usually impossible to decide whether the perforation is due to the sudden breaking-down of an acute, or to the gradual decomposition of a chronic tuberculous focus, or merely to rupture of an emphysematous alveolus. In most cases the duration of pneumothorax, whether it end in recovery or in death, extends over days or weeks, so that the local condition of the tissues at the time the escape of air occurred cannot be satisfactorily determined.

One thing is certain, namely, that chronic, disseminated bronchopneumonia, which is more often associated with dense adhesion of the pleura than is the simple caseous lobar pneumonia, more rarely leads to pneumothorax because rupture does not occur in the presence of dense pleural adhesions except under very special circumstances, such as an unusually rapid break-down of a gangrenous focus at the periphery, with secondary necrosis of the pleura.

I twice observed the occurrence of **pneumothorax in whooping-cough;** both cases occurred in small children and both ended fatally. The cause of the pneumothorax was not definitely determined, as there were no autopsies. It is probable, however, that in one case the complication was due simply to rupture of an alveolus by the unusually severe and persistent paroxysms of cough, while in the second case the accident may have been due to necrosis of a bronchopneumonic focus, physical signs of which were present.

In abscess of the lung following pneumonia, pneumothorax is not a common complication, because the abscess, as a rule, soon becomes encapsulated.

It may be incidentally mentioned that pneumothorax may also be produced by the rupture of echinococcus cysts.

Pneumothorax due to an **ulcerating tumor of the thoracic wall,** to **peripleuritic abscess,** or to **caries of the ribs** is very rare, because in all such cases the development of the condition is accompanied by obliterating processes in the pleura. It is somewhat more frequent as a complication of rupture of a **mediastinal suppuration,** rupture of an **esophageal carcinoma, tuberculosis, pericarditis,** and **ulcerative processes in the intestinal tract.** A few cases are produced by direct suppuration or ulcerative processes in the larynx or esophagus by a foreign body.

Pneumothorax due to external **traumatism,** stab and gunshot wounds, etc., does not fall within the province of this article.

III. MECHANISM OF PNEUMOTHORAX.

Notwithstanding the large number of available investigations, including especially the older clinical and experimental investigations by Wintrich and the more recent ones by Weil, the mechanism involved in the production of large accumulations of air in the pleura appears to us still to require some elucidation. When it is considered that after an empyema operation, in spite of the free opening of the intercostal space and in spite of complete and persistent retraction of the lung, the two surfaces of the pleura may become completely adherent within a few days, or even a few hours, providing the lung is capable of expanding and the healing process has not been disturbed by improper methods of treatment, such as forced irrigation, or by destructive processes in the lung—when it is further considered that under the most varied conditions as regards the tissues and character of the opening we may have at one time pneumothorax with high pressure, and at another time slight or barely demonstrable escape of air; and, finally, since these clinical investigations are further confirmed by experiment, it appears evident that the pathogenesis of pneumothorax is not so simple as to be explainable solely by the mechanical conditions without regard to the local or general tonus, that is to say, the mechanism at the site of perforation and the changes in the innervation of the entire respiratory apparatus.

If, after dividing the skin, the intercostal spaces of an animal are perforated with a blunt cannula and an attempt is made to introduce air or fluid under moderate pressure, an enormous resistance is encountered. It is often quite difficult, and, if the animal is not anesthetized, even impossible, to produce pneumothorax or hydrothorax; or, in other words, to introduce a foreign medium into the pleural cavity at the expense of the pulmonary volume. Even when a large opening is made, the lungs, if the animal is not anesthetized, do not collapse to the same extent as is usually observed in the cadaver.

It is evident, therefore, that the living organism possesses an apparatus which reinforces the coaptation of the pulmonary and costal pleura; and the function of this apparatus is the more perfect, the better the state of the respiratory apparatus itself. It seems to be almost as difficult to draw apart two glass plates as to separate the two healthy pleural surfaces by a foreign agent, as the wound appears to become at once hermetically closed by vicarious activity (increased expansion) of parts at some distance from the wound.

The same thing is true of wounds in the pulmonary pleura, the healing of which is even facilitated by the fact that the first escape of air causes the parts immediately surrounding the incision to collapse, and thus acts as a kind of tamponade which prevents the further entrance of air into the peripheral portions, and therefore also interferes with escape of the air into the pleural sac. In the same way the opening is immediately occluded in cases of hemorrhages, extravasations of serum, or formation of fibrinous or inflammatory products, unless there happens to be a gaping bronchus, not to speak of the intrinsic elasticity of the tissues.

It is obvious that the compensatory dilatation of remote portions, as explained above, is in direct proportion to the intensity of the local intrapleural pressure which affects contraction of the lung in the immediate vicinity of the fistula. This local external expiratory stimulus acts as an inspiratory stimulus for those parts which are not under the direct influence of the foreign stimulus; or, in other words, any stimulus which is capable of producing collapse of one portion of the lung reflexly induces expansion of adjoining portions, and thus the diminution of volume is compensated for as much as possible.

Whether we have to do with a pneumothorax, with an external, internal, or combined form of fistula, the inflation of the pleural cavity during inspiration necessary for any extensive accumulation of air will require more favorable conditions for the entrance of air into the pleural cavity than for the expansion of the lung, which is the only factor concerned in maintaining constant direct contact between the pleural layers, and thus hindering the escape of air.

A cavity can be produced between the two layers of the pleura only when the lung is incapable of expanding, and the pulmonary pleura during inspiration becomes separated from the costal pleura, when the pleura or thoracic wall during inspiration fails to undergo uniform concentric shrinking, or the abdominal viscera are unable to follow the movements of the diaphragm. In other words, if there is an adequate communication between the lung and the pleural cavity, the pleura will be filled both during inspiration and during expiration; while if one or the other of these factors predominates, an increase of the accumulation of air can occur only in one of the two phases.

In any case, whether there is an open fistula or one that can be closed, there must be some disturbance in the relations between the lung and the thoracic wall and some alteration of the tissue tonus to facilitate the entrance of air.

It seems to us, therefore, that the nature of the fistulous opening, the so-called valve-formation, on which a special subdivision into open and closed valvular pneumothorax has been based, is of less importance than the condition of the respiratory apparatus itself—that is to say, the nature of the tissue and its power of performing accurate work by which a movement of the parts in the same direction is produced during respiration. Paradoxical as it may sound, the real provisional closure does not take place at the site of perforation. It is owing to the behavior of the other portions that, in spite of the existence of an opening, little or no air can escape through the pulmonary or costal pleura. The compensatory change in the volume of the lung is such that there is no room for the air and no increase of the available space in the pleura.

Maintenance of the normal pressure relations will, of course, depend on the size of the opening, the rigidity of its walls, the elasticity of the lung tissue in the immediate vicinity, and the irritability of the reflex apparatus.

When there is a direct communication between a large bronchus and the site of perforation, or when a small rigid bronchus gapes at the surface of the pleura, the mechanism is of course unable to act, and the fistula cannot be securely closed because the pulmonary air communicates with the pleural cavity by means of a relatively large canal with rigid walls. In this case the pressure of the air on the opposed surfaces of the pleura—in the capillary space—must be greater than on the bronchial side of the other portions of the lung, where it is diminished by the full extent of the elasticity and of the tonus of the lung (which is capable of retracting), and air will therefore enter the cavity at each inspiration, and possibly also during expiration, if the air-pressure in the bronchial system is greatly increased during this phase.

In the same way the costal pleura becomes separated from the pulmonary pleura, and air is drawn into the cavity when there is any diminution in the tonus of the lung (which is not to be confounded with impairment of physical elasticity), any alteration in the lung tissue offering an increased resistance to the internal air-pressure which expands the lung, and, finally, during every inspiratory movement. The greater the rigidity of the pulmonary tissue, the larger will be the amount of air drawn into the pleural cavity. If, owing to the insufficiency of the internal pressure (the intrapulmonary column of air), the expansion of the lung in the vicinity of the fistula fails to keep pace with the escape of air from a bronchus opening at that point or from any other opening surrounded by unyielding tissue, every facility is afforded for the free access of air to the pleural cavity until the amount of air passing through the fistula becomes less than that which enters and inflates the organ.

A number of other factors may of course also enter into the question and modify the ultimate result, but a satisfactory general idea of the mechanism and the variety of possible combinations may be obtained from the above explanation.

It is to be remembered that the pulmonary tissue is under the influence of an active tonus and that the inspiratory expansion does not depend merely on the difference between the internal and external air-pressure, as, for instance, in an inflated rubber ball, but chiefly on the relaxation of the tonus. It is analogous to the diastole of the heart, where the dilatation of the cavity is not effected by the pressure of the blood, since the blood-current merely fills a capillary space formed by relaxation of the tonus. In the same way, during the respiratory expansion of the lungs inflation of the organ is performed purely by the variations in tonus without any actual distention by increase of air-pressure.

The pressure on the pulmonary tissue must not be greater at the end than at the beginning of a full inspiration, paradoxical as this may sound, and notwithstanding the fact that our manometric methods indicate a slight (negative) difference in the trachea. A true physical distention could be effected only by abolition of the vital tonus. The muscles which dilate the thorax do not contract until the lung begins to relax; they then contract in proportion to the relaxation of tonus, and thus bring about filling without distention. The muscles relax again only when the tonus of the lung is restored and gives the signal for expiration. Both processes are controlled by a reflex innervation, a far-reaching synergy, so as to preserve precision in the co-operation of the various parts of the apparatus (see page 915).

To recapitulate, the greater the differences in pressure in the system of canals communicating with an internal pleural fistula and in the other portions of the internal surface of the lung which still retain their normal power of retraction, the freer the entrance of air into the pleura.

At the mouth of the fistula, especially if it is surrounded with rigid walls, the pressure between the layers of the pleura during the respiratory pause falls from full atmospheric pressure to zero; during inspiration the pressure falls to a negative point corresponding to the degree of rarefaction possible; during expiration the pressure falls to a level determined by the (positive) increase of pressure in the bronchi and the pressure within the pleural cavities.

The pressure within the pleura, provided there are any respiratory movements at all on the affected side, making it possible for an air-containing space to be formed, depends on the extent of this space and the amount of the escaping gas.

Outside of the fistula, where the air is confined between the pleura and the lung, the pressure depends on the tonus of the lungs and of the chest-wall. The pressure on the inner surface of the pleural cavity is diminished by an amount equal to the degree of pulmonary tonus and the elasticity and power of expansion of the chest-walls.

Since, as has been mentioned, the difference between the pressure in the fistula and the intrapulmonary pressure in other portions of the lung is proportional to the rigidity of the tissues surrounding the fistula, and the inability of these tissues to follow the traction of the inspiratory muscles, the air escapes in the line of least resistance—that is, toward the opening—with a rapidity proportional to the width of the bronchus which communicates with the fistula and the functional impairment of the remaining pulmonary tissue. The greater the loss of elasticity in the diseased lung, the greater will be the accumulation of air within the cavity; hence the accumulation of air is greatest in phthisis and other marked diffuse alterations of the lung tissue, and is least in a normal lung. It is obvious that in emphysema the amount of escaping air cannot be very great because of the diminished power of the lung to retract; in other words, the strength and rapidity of the air-current in the system of canals communicating with the fistula may, under certain conditions, be greater than the strength and rapidity of the fall in pressure which enables the lung to relax and adapt itself to the pull of the thoracic wall.

The air cannot return during expiration because no expiration can take place in those portions of the lung where the true inspiratory dilatation of the tissue, either partial or total, is replaced by the entrance of air into the pleura. Even when the fistula is constantly open, the mere sinking-in of the chest-wall cannot bring about a sufficient increase in pressure to expel the air from the pleura into the lung against the more powerful mean intrabronchial pressure. To effect this would require the exertion of active muscular expiration, which, as observation teaches, is seldom performed.

Even granting an increase of pressure in the pleural cavity during expiration, the air cannot always return unless the mechanism of innervation and the normal condition of the tissue undergo a complete change, since the conditions in the fistulous tract and in the pleural sac are now the exact reverse of what they were before. The internal air is more than ever under the influence of the healthy lung which functionates more actively than under normal conditions, and therefore raises the positive pressure within the bronchial system. The air within the pleural sac, on the other hand, is under the influence of the expiratory pressure of the diseased side, which is usually weakened;

hence in either case the relative increase of the intrabronchial pressure, which is most pronounced in the fistulous canal on account of its rigid walls, offers considerable resistance to the return of air from the pleural cavity during expiration. Under favorable conditions the escape of air may even be most marked during expiration.

The importance of the expiratory pressure is shown by the fact that the expansion of a lung after empyema operation is chiefly, or at least very largely, effected by the expiratory pressure. In this case, however, the conditions are reversed: the chest wound, being wide open, allows the air to escape freely, so that the increase in the mean expiratory pressure, especially when the insufficient action of the expiratory muscles on the diseased side is considered, results chiefly in expansion of the lungs and the expulsion of the exudate of air through the large outer opening; in other words, the mean intrabronchial pressure (since the pressure in both lungs becomes neutralized in the bronchi), and therefore the intrapulmonary pressure in and around the fistula, may, under the influence of certain tissue changes in the lung and of certain changes in the tonus, be greater than the pressure on those regions where the tonus of the normal tissue resists expansion or than the intrapleural pressure in this region and in the entire diseased lung.

This applies to all cases in which the vital power of the lung tissue has suffered; it does not apply where the lung tissue is normal, where there is no wide canal with rigid walls and the communication is effected merely by a small circumscribed tear in the tissue. Whether the fistula be open, or manifest a tendency to close completely or by the so-called valvular method, the filling of the cavity and the rise of pressure in the pleural space depend less (or certainly not more) on these local conditions than on the condition of the lung tissue in general. Under favorable conditions the fistula must, owing to the affinity of the tissues, eventually become closed by the vicarious activity of neighboring organs; that is to say, inflation brought about by the necessary relaxation of the tonus and adaptation of the parts effects a kind of tamponade about the fistula, diminishing the movement of the air within the fistula to such an extent that the healing process has a chance to begin restoring the continuity of the tissue. It goes without saying that the presence of fluid or solid exudates may modify the conditions just explained. And these products of inflammation also modify and hasten to a certain extent the closure of the fistula, in accordance with what has just been said.

Weil distinguishes between complete obstruction or obliteration of a valvular pneumothorax, which he calls organic occlusion, and the partial obstruction produced by expiratory pressure, which he calls mechanical occlusion. This distinction cannot, in our opinion, be carried out in practice, nor is it always justified in theory, as the escape of air does not by any means depend solely on inspiration and is not always inhibited by expiration, as is shown both by the above explanation and by experience.

The positive pressure in the thoracic cavity may, of course, be considerably increased in elastic chest-walls, in failure of the lung to expand, and when, owing to the relaxation of tonus, the neighboring organs retract. If under such conditions an external fistula is made, the air will even escape with a hissing noise.

Such a rise in pressure is always a sign of marked primary disturbance in the parenchyma of the lung, and indicates that the chest-walls or other boundaries of the pleural cavities are unusually yielding as compared with the abnormal rigidity of the lung tissue. When the lung fails to follow the contraction of the powerful chest muscles and the current of air at the fistulous opening is stronger than the air-current in other portions of the tissue which expands the lung, or, more correctly, makes it possible for it to expand, a greater amount of air will force itself between the layers of the pleura until the fistula becomes tightly closed by some form of tissue alteration. But how can such closure result if a constant current of air prevents any form of superficial healing unless collapse and hepatization take place?

Closure of the fistula by healing of the tissues would, of course, be extremely desirable under these very conditions, as the vital processes which normally bring about closure have suffered an impairment of function.

But unfortunately, in addition to the insufficient power of regeneration of the tissue, other unfavorable factors are here at work; for the failure of the lung to expand must, owing to the diminution in the respiratory surface, be equalized by an increase in the reflex activity of the muscles of inspiration. But an increase in the work of these muscles, instead of expanding the lung, merely results in augmenting the escape of air into the pleural cavity. After a while the insertions of the voluntary muscles approach each other more and more, and contractures are formed which interfere with inspiration, while the overdistended intercostal muscles also undergo a species of permanent reflex contraction which only tends to increase the positive pressure without bringing about recovery—that is, effecting the return of the escaped air into the bronchial system.

Various observers, among whom we shall mention Leyden, Peyrot and Weil, have determined the air-pressure by manometric methods. By a modification of the methods in use Weil attempted to investigate the differences in pressure between an open and a closed pneumothorax, and, as has been mentioned above, between organic and mechanical closure. But the figures obtained by the various authors diverge so widely that I do not believe this method is of any value in practical work.

I have found that in experimental pneumothorax different results may be obtained in different portions of the pleura, and I believe that in pneumothorax due to morbid processes in the lung even more widely varying results may be obtained.

To demonstrate pneumothorax in the cadaver, the skin over the upper anterior portion of the diseased half of the thorax is dissected away from the middle toward the side. The space between the skin and the muscles is then filled with water, so that when the intercostal spaces are opened the gas-bubbles rise to the surface and become both visible and audible.

There are, of course, numerous sources of error in this experiment, because the expulsion of the gas depends on the power of retraction of the dead tissues, and the air-pressure, which was positive during life, may after death become diminished or entirely abolished by cooling of the parts, rigor mortis, and other as yet unknown processes (diffusion, absorption?), and, finally, because the gas that is demonstrated may not have been originally present and may be due to postmortem decomposition processes in the pleural fluid.

IV. CAUSES OF THE EXUDATION.

The accumulation of air may, as has been mentioned above, be combined with an accumulation of fluid. The causes for the production of the latter have never been satisfactorily set forth, and it is difficult, without forcing the argument, to explain the fact that not a few cases of pneumothorax with an internal or external fistula run their course without giving rise to any irritation of the pleura or any demonstrable accumulation of the fluid.

Are we to assume that in such cases the air that entered the pleural cavity was quite free from germs, while in others some material which acts as a cause of irritation, such as bacteria or corpuscular elements, finds its way into the cavity along with the air? This explanation seems the more probable since the ordinary course of events after the opening of an uncomplicated abscess in the external skin, or even in the thoracic or abdominal cavity, tends to show that the mere entrance of atmospheric air cannot with any degree of positiveness be regarded as the cause of inflammatory or even putrid processes in the pleura and peritoneum.

We may, therefore, conclude that the kind of inflammatory reaction in the pleura depends on the degree of irritability (disposition) of the membrane at the time of invasion; in other words, on the nutrition of the entire body and of the lung itself. This, of course, does not apply where perforation has taken place and large masses of material that act as strong irritants to the tissue, such as the putrid contents of bronchiectasis, have made their way into the pleura.

The normal pleura, in my opinion, does not appear to become inflamed by mere exposure to the air, and even an inflamed pleura,—such, for instance, as a tuberculous pleura without any fluid or plastic exudate—is not affected by the entrance of air to the extent of producing any marked exudation. I have frequently known pneumothorax to persist for some time in cases of tuberculosis of the pleura without any exudation whatever.

In tubercular disease of the lung itself, which is so frequently followed by empyema, and in all forms of tuberculosis of the respiratory apparatus, the pleura is probably always in a highly irritated condition, so that the entrance of air or even of the contents of a cavity is not necessarily followed by an increase in the irritation sufficient to account for a purulent exudation or the subsequent putrefaction of the exudate.

We may, therefore, assume that the pleura under these conditions is an especially bad medium for the growth of organisms which produce inflammation, or else that the large numbers of pus-corpuscles and other migratory cells in the tissue temporarily destroy the micro-organisms that enter the pleura through a perforation.

It is also a striking fact that it is comparatively rare to find pneumothorax combined with other than a serous exudate or a combination of serous and purulent effusions. And the fact that even in tuberculous pneumothorax the exudate is relatively seldom—that is, in view of the great frequency of these cases—putrid from the beginning also tends to show how slight a tissue irritant the air must be. The exudate, as a rule, becomes purulent only after repeated aspiration, when the lung tissue begins to break down or the patient develops high fever and great loss of strength.

As a result of extensive clinical observations, it may be laid down as a rule that the entrance of air in a *normal pleura* is usually not followed by exudation; in a *slightly irritable pleura* by the formation of a serous exudate; and only in a *very irritable pleura* by purulent or putrid decomposition. Atmospheric air, even when badly vitiated, appears to act only as a very slight irritant to the pleura providing the latter is not very irritable, for I have not infrequently seen both uncomplicated and complicated cases of pneumothorax run an unexpectedly favorable course under the most unfavorable surroundings.

According to Senator, the spongy tissue of the lung acts as a kind of filter for the intrapulmonary air, and the carbonic acid gas contained in the lung also plays an important part in preventing inflammatory action by the power it possesses of disinfecting the air.

It has been pointed out by Wintrich how easily animals appear to tolerate artificial pneumothorax, and how rapidly large masses of air may undergo resorption without any inflammatory reaction.

It is shown by the experiments of Szupak that the healthy pleura in closed pneumothorax is capable of absorbing very considerable amounts of air. Carbonic acid gas and oxygen are absorbed with the same rapidity as atmospheric air, while only a part of the nitrogen present becomes absorbed in the same time. In artificially produced sero-pneumothorax (the author was unable to produce a purulent one) the pleura was found to possess considerable power of absorbing air.

These investigations, therefore, prove that the atmospheric air in itself cannot be regarded as an irritant, and that the suppuration which undoubtedly does follow the entrance of air must be attributed to other causes than to the mere irritation of the air.

A. Fraenkel's view that the favorable course in cases of pneumothorax depends on an especially low percentage of germs in the entering air cannot, therefore, be regarded as positively proved, nor can we admit that early closure of the fistula is alone responsible for a favorable course, although, as has been stated above, we do not deny that early closure of the fistula is a very favorable and much-to-be-desired event, since it always indicates that function of the entire respiratory apparatus is relatively normal; in other words, that a large part of the tissue has preserved its integrity.

Nor do I agree with A. Fraenkel when he reasons from his investigations that whenever the exudate of a pyopneumothorax produced by rupture of a cavity is found to contain only very few tubercle bacilli and no pyogenic micro-organisms, the pus from the cavity must have originally contained only a minimum quantity of tubercle bacilli, as I cannot conceive how it is possible that the contents of a perforating cavity should be entirely free from micro-organisms. It seems to be much more plausible that the interesting absence of bacteria in such cases may be explained by the assumption to which we have already referred that the pleura under certain conditions is a very bad medium for the growth of micro-organisms.

The **extent of the exudate** in pneumothorax varies like that of any other exudates produced in other forms of pleurisy. The fluid contents of the pleura may amount to many liters and, as has been pointed out by Senator and Weil, bilateral exudates may occur, whereas bilateral pneumothorax is an exceedingly rare event.

It is to be remembered that the exudate is always much more abundant than one is led to expect by the results of percussion and other methods of examination. This is due to a variety of causes, chief among which is that, owing to the great resonance of the cavity, the dulness is relatively small, and, in addition, the pressure of the air on the chest-walls renders them much more yielding and more capable of taking up fluid, so that even a large exudate does not rise to the corresponding level.

According to many authors (Wintrich, Lebert, Saussier, Weil), the perforation in tubercular pneumothorax occurs much more frequently on the **left side.** The cause of this peculiarity is found by Weil in the asserted more rapid development of tuberculosis in the left lung and in the effect of changes in the volume of the heart on the border of the lungs, whose respiratory activity, besides, is greater than normal; that is to say, in processes which prevent or at least delay the formation of adhesions.

While I can confirm the belief that pneumothorax is more common on the left side, I am unable to offer any explanation. In my observation the majority of cases of pneumothorax, excluding those due to phthisis and traumatism, affected the left side. [In Morse's series the pneumothorax was on the left side in 29 out of 44 tubercular cases. According to Rose, 14 cases of bilateral pneumothorax have been reported so far.—Ed.]

It is not always easy to find the perforation in the cadaver unless a bronchus has been permanently laid open or the thin wall of a cavity has burst and death has occurred before the opening has healed over. When opportunity has been afforded for the formation of adhesions, it is very difficult to find the site of perforation which usually is in the shape of a lentil, more rarely a fissure.

A perforation may easily be simulated in lungs containing many cavities which have become closed by firm adhesions, as it is not always easy to avoid tears in the attenuated portions of the lung even when the greatest care is exercised in taking out the thoracic organs. It is better, therefore, to demonstrate the existence of an opening by immediately making a pocket, in the manner described, and if necessary inflating the lungs through the trachea.

V. SYMPTOMATOLOGY.

1. GENERAL CONSIDERATIONS AND INSPECTION.

In fairly vigorous patients the occurrence of pneumothorax is usually attended with unmistakable and very severe symptoms. If the patient is feverish and debilitated and already suffers from dyspnea, the onset of the disease is often disguised to such an extent that even a considerable escape of air into the pleural cavity is discovered only accidentally or after a very searching examination.

In the first class of cases it appears very clear that the intensity of the symptoms depends not on the amount of air within the cavity, but solely on the action of certain pleural reflexes. There seems to be a kind of *shock* or very distressing condition resembling angina pectoris produced by certain abnormal sensations in the course of the vagus or the intercostal nerves, due to the altered mechanical conditions in the thoracic cavity, possibly the greater tension of the pleura, or the modified mechanism of respiration.

Both the subjective and objective symptoms are much intensified when the escape of air is accompanied by the feeling of something giving way in the chest. In such cases there is usually pronounced *collapse* with marked dyspnea, sometimes with orthopnea, small, almost imperceptible pulse, coldness of the extremities and of the face, cold sweats, and marked fall in temperature, as in thrombosis of a large pulmonary artery.

These symptoms, which are directly produced by reflex irritation of the vagus, of course depend on the general reflex irritability of the organism, the patient's general and mental condition, the degree of diminution of the respiratory surface, the pressure of neighboring organs, and the power of compensation. They therefore do not possess any direct value for the diagnosis or prognosis of pneumothorax.

After these symptoms of shock have subsided, the following condition is usually observed, especially in cases of so-called valvular pneumothorax, where the air may accumulate slowly or rapidly: The breathing is accompanied by vigorous play of the alæ nasi and of the muscles of the neck, the patient tends to assume an erect position, and usually protrudes the healthy side somewhat so as to diminish the circumference of the diseased half of the chest.

The **enlargement** of the affected side is often visible in the very beginning, the intercostal spaces are tense and very sensitive, the lower portions of the chest are often immovable, while the corresponding portions of the other side show considerable increase in the respiratory movements.

The respiratory movements in the various forms of pneumothorax have been experimentally investigated by Krebs, who found that open pneumothorax was attended by an increase both in the frequency and depth of the respirations, while in closed pneumothorax the frequency was decreased and the depth increased. If, however, more than a certain quantity of air is injected, the depth of the respiratory movements is also diminished, so that eventually the respiration becomes even more shallow than normal. These results have been subsequently modified in various respects by the investigations of Blumenthal. No general statement can be formulated on the basis of clinical observation in regard to depth and frequency of respiratory movements in closed or open pneumothorax. In most cases the nature of the breathing is of no value in the differential diagnosis.

The **position of the apex-beat** varies in different cases. In right-sided pneumothorax it is said to be displaced to the left; in left-sided pneumothorax the pulsations of the heart are said to be stronger and the apex-beat is displaced to the right. This, however, is found to be true only in a small number of cases; much more frequently the apex-beat is entirely absent, an indistinct wavy bulging is seen in different portions of the cardiac region, or a slight thrill or rubbing sound is felt over this area. It is easy to understand why this should be so, since in right-sided pneumothorax the left lung undergoes complementary expansion, while left-sided pneumothorax often produces a considerable depression of the diaphragm, resulting in dislocation of the heart downward, so that only the pulsation of the right ventricle is felt in the epigastrium, as in the highest grades of emphysema. Besides, as the accumulation of air often occupies the neighborhood of the heart and causes a bulging of the chest-wall in this region unless the patient is in the erect position, the pulsation of the heart cannot, of course, be increased, and it may be difficult even to recognize the normal pulsation of the organ. The lung in such cases is always displaced toward the clavicle, and does not extend as far posteriorly as usual.

Pneumothorax may also be combined with pulsating pleurisy. It is found almost exclusively after pneumothorax of the left side, and in the great majority of cases is also accompanied by empyema necessitatis (see page 896).

No general statement in regard to the **temperature** in pneumothorax is possible, as it all depends on the primary trouble. As a rule, the temperature falls during the first hours or days, as the occurrence of pneumothorax is often followed by collapse and shock; later the primary disease reasserts itself, and in general the fall of temperature at the time the air escapes is inversely proportional to the height of the fever that had been present before. As the exudate increases the fever rises more and more, and often assumes a *hectic character;* in putrid exudates *subnormal temperatures* are often observed.

Occasionally the formation of a pneumothorax has been observed to be followed by repeated attacks of urticaria, as after the rupture of echinococcus vesicles in the peritoneal cavity (Senator and Gerhardt).

[Kaienbeck studied a case of closed left pneumothorax with the **fluoroscope.** There was a vertical movement of the upper surface of the fluid with respiration, probably because in this disease the diaphragm is convex below and therefore moves upward in contracting. There was pulsation, due to the direct transmission of the cardiac impulse, not to propagation of the impulse from air to fluid, since the pulsation ceased when the patient lay on the left side and the heart was entirely surrounded by air. This is a valuable proof of the correctness of Traube's view that the pulsation of the empyema is due to direct transmission of the impulse from the heart.—Ed.]

2. PERCUSSION.

Unless the chest muscles are spasmodically contracted the percutory note, even when the accumulation of gas is only moderate, is so strikingly **loud and deep** that one is at first apt to think that there is dulness over the other half of the thorax. The tone almost never has a distinctly tympanitic character even in moderate pneumothorax, nor do we ever hear a cracked-pot sound when the chest muscles are completely at rest, if the tension of the chest-walls is at all marked. In left-sided pneumothorax the *cardiac dulness* is usually diminished or altogether obliterated.

True dislocation of the heart by the mere accumulation of air even in the left pleural cavity occurs much more rarely than in pleural effusions. There is merely a diminution of the dulness or it becomes entirely masked by the loud pulmonary resonance. In right-sided pneumothorax the *liver dulness* often disappears, or more frequently the organ becomes displaced. Rotation of the organ about its axis is, of course, also met with, as has been described in connection with pleurisy (see page 838), but the conditions necessary for its production cannot be predicted for all cases, as the phenomenon depends on the action of the diaphragm. One of the most positive signs of right-sided pneumothorax is, of course, the sudden and marked **displacement of the lower border of the liver,** which can sometimes be felt at the level of the umbilicus in the middle line of the abdomen. An equal degree of displacement is rarely noted in pleurisy. This difference in the dislocation of the liver in pneumothorax and fluid exudate depends on the suddenness with which the alteration takes place. It is probably due to the fact that in pleurisy, which in general develops gradually, the chest muscles and the diaphragm participate in the inflammatory process and become weakened and more yielding, while pneumothorax, from the suddenness of its development, elicits a reflex antagonistic contraction of the muscles of the entire circumference of the thorax, thus increasing the tension of the thoracic cavity and facilitating the enlargement of the thorax downward.

The percutory phenomena described are also observed on the dorsal surface, and, owing to the extreme intensity of the sound, it frequently happens, as has already been mentioned, that even large accumulations are overlooked, or at least give rise to error in regard to their size.

The manner in which the abnormal alterations of sound are produced need not be entered into here. It may be remarked, however, that the abnormal loudness and depth of the tone are produced not by the tension, but by relaxation of the tonus in connection with diminished muscular action. The greater the accumulation of air, and therefore the tension, the more shallow the sound, although the loudness still persists until the highest degree of tension (physical distention) is produced, when the sound gradually begins to lose its loudness.

Although the physical conditions are apparently simple, they are materially modified by a whole series of processes, and the percutory phenomena are therefore usually different from those observed in meteorism of the intestinal tract.

Tympany occurs only when the pressure of the air within the pleural cavity is diminished; that is, when, under the influence of active tonus, the lung is retracted, and also when the chest muscles are relaxed and a larger amount of air accumulates on the anterior surface of the thorax beneath the clavicle. Under such conditions the diagnosis may be exceedingly difficult, as it becomes necessary to differentiate the sound from the so-called Skodaic resonance which is observed in moderately large pleural effusions (see page 859) and from the percutory phenomena that are produced by the presence of large cavities and are usually associated with bronchial breathing. The most trustworthy sign in such cases is the *dulness over the infero-posterior portions of the thorax*, which practically settles the diagnosis, for it is only when there is a marked dulness in the back due to the presence of a pleuritic exudate that the tympanitic note below the clavicle can be regarded as a positive sign of retraction of the lung and as a true Skodaic resonance.

In the diagnosis from large **phthisical cavities** the general condition of the individual, the history, the other physical signs, and the examination of the sputum must, of course, be taken into account.

The various modifications of the percutory note with change in the position, although apparently characteristic, do not appear to me to possess any great diagnostic significance, as they usually depend more on alterations in the chest-wall than on displacement of the fluid.

It has been pointed out by Biermer that, owing to the change in the diameter of the pleural cavity, the pitch must be higher in the dorsal than in the erect position, but the exact opposite has not rarely been noted, both by myself and by other observers, and while the changes in the sound described by Biermer may be more common than any other modification, they cannot be said to give any material aid in the diagnosis.

The statement by Gerhardt, that in open pneumothorax the pitch is higher when the mouth is open than when it is closed (as in Wintrich's phenomenon in open cavities), may, as has been shown, be explained by the fact that the act of opening the mouth is accompanied by increased tension of the chest-wall, since most persons involuntarily draw in the breath when they open their mouths.

As far as my observations have gone, the distinct displacement of the dulness incident to change in position more frequently depends on a change in the volume of the lung accompanying the change of position than on the displacement of the mass of air or fluid itself. As the patient changes his position he feels a need of breathing more deeply; the lung accordingly contains more air than before and the increased resonance extends over a larger area, so that the upper border of the dulness becomes displaced downward.

It is, of course, not to be denied that the modified note in many cases is due to the change in the level of the exudate, especially when atelectasis can be excluded. If, therefore, the lungs are positively known to be infiltrated or consolidated, a distinct change in the note is a more important sign than when the lungs contain air, since under such conditions a change in the dulness can only be due to a displacement of the boundary between fluid and air.

In pronounced cases of pneumothorax, in which the diseased half of the chest is absolutely immovable, **pectoral fremitus** may be everywhere absent. If it is distinctly present in one spot or over a larger area, one of two conditions is present: either there is a large bronchus at the surface of the lung, or the lung is retracted and infiltrated or adherent to the costal pleura, and therefore affords an especially good conducting medium for the sound of the voice at the particular spot in question. When the air in the pleura is under a certain pressure, and particularly when the thoracic wall is much distended, the vibrations of the voice are very imperfectly transmitted.

3. AUSCULTATION.

Auscultation frequently yields an absolutely negative result. The **respiratory sounds** are either absent altogether or greatly diminished in intensity and murmurish. Bronchial breathing occurs only in portions of the lung the seat of infiltration or other pathologic change, or where the lung tissue, in spite of the pressure of the air, is not completely compressed, or in the neighborhood of larger bronchi. As a rule, the remnant of vesicular breathing has a slight metallic or amphoric quality, the intensity of which depends on the strength of the respiratory movements and on the size and shape of the vibrating cavity. The point of greatest intensity of the metallic (bronchial or undefined) sound is below the clavicle, although it may be heard quite distinctly in the lateral wall or the supraclavicular space; in rare cases it is equally well heard over the entire half of the thorax. Generally speaking, however, this variety of metallic sound—that is, distinctly perceptible vesicular murmur with a metallic quality—is rare; and as the results of auscultation are always unsatisfactory on account of the lack of respiratory movement in the lung, other methods must be employed to reinforce the sound-waves of the respiratory murmur, and thus to accentuate the metallic resonance of the thoracic cavity. Hence if pneumothorax is suspected, the air in the bronchial system must be thrown into more active vibration. Either the patient is asked to cough, which usually brings out the metallic phenomena, or the stethoscope is applied to the chest while percussion is performed by tapping the pleximeter with the handle of the plessor or with some other solid body (*pleximeter percussion*, after Heubner and Leichtenstern). In this way overtones are produced which possess a splendid metallic quality.

By carefully shaking the patient a loud metallic **splashing sound,** *succussio Hippocratis*, can be produced which is often audible at some distance. It appears to me doubtful whether this splashing sound is always produced by a mixture of air and fluid within the pleural cavity. Under favorable conditions for resonance it may also be due to other causes; for in many cases a splashing sound produced in the stomach or abdomen acquires a strong metallic element as it passes through the resonating pleural cavity, just as râles and heart sounds under especially favorable conditions for resonance acquire a metallic character in the distended organs of the abdomen.

The so-called **falling-drop sound** (*tintement métallique*, metallic tinkling), after Laennec, is very interesting from a theoretic standpoint and possesses some diagnostic value. It is a distinct, musical metallic tinkling which, in my opinion, resembles the sound of a glass harmonica or the sound produced by agitating a metal pitcher containing water more than it does the sound of water dropping into an empty cask. This peculiar tinkling with its long after-tone cannot, as might be supposed, be due to the falling of a drop of fluid, since there is nothing to indicate the possibility of such a thing happening in the pleural cavity. It is much more probably due to a râle produced in some portions of the lung which acquires a metallic character owing to favorable conditions for resonance. Similar noises are not infrequently heard in the intestinal tract, though they do not always possess the same strongly musical quality which characterizes the phenomenon in pneumothorax.

Another sound that belongs to this class of phenomena is that first described by Dance and Beau; it is heard during inspiration, and consists of a comparatively long-drawn-out gurgling or metallic sound with a coarse vesicular quality. It has recently received the name of **water-pipe sound** (*Wasserpfeifengeräusch*, Unverricht) on account of its resemblance to the noise produced by the air-bubbles as they are sucked through the water in smoking a tschibuk or hookah. Riegel calls it a lung-fistula sound.

Unverricht heard the sound in two cases of open pneumothorax after a few syringefuls of exudate or air had been evacuated, and assumes that after the pressure within the fistula, which up to that time had been mechanically closed, is relieved by the partial evacuation, a certain amount of air is drawn through the fluid from the lung at each inspiration. Riegel also observed the phenomenon during inspiration when the pressure had not been altered by operative evacuation but by the spontaneous expectoration of a large amount of exudate through the bronchi.

I do not believe that this sound is any more characteristic of pneumothorax in general than it is of valvular pneumothorax (open pneumothorax). Weil has pointed out that a similar sound may be produced in large cavities in the lung itself without the presence of pneumothorax, and I believe that it is always produced when coarse râles are present and the conditions for metallic resonance are unusually favorable.

No more favorable conditions for the production of such phenomena can be found than in the moderately retracted lung of pneumothorax, since the passage of air through the larger bronchi is still possible and the pneumothorax offers an ideal resonating chamber for the metallic sounds. According to this view, the so-called water-pipe or lung-fistula sound cannot be produced if the lung is compressed and the air cannot enter the large bronchi; this shows why it is absent in total compression of the lung while it may be produced by any lowering of the pressure from the evacuation of the exudate either by spontaneous expectoration or operative aspiration. Indeed, it must be present whenever the production of coarse râles is made possible by the re-establishment of a slight air-current through the larger bronchi of the corresponding lung, as these râles of course acquire a striking metallic resonance in the air-filled pleural cavity.

Granting that this explanation is correct, the sound will be more frequently heard during inspiration than during expiration, since inspiratory râles must be more frequent in a lung compressed by pleural exudate and in so-called mechanical valvular pneumothorax.

There is, however, nothing to prevent the occurrence of expiratory or even continuous metallic râles under such conditions. They will usually be of the large bubbling variety, provided always that the air has access to a large bronchus in a certain phase of the respiratory rhythm. Small moist râles of the same quality are, of course, more rare, because a moderate evacuation will rarely open the smaller bronchi sufficiently to admit a current of air. It follows from all that has been said that the sign under discussion cannot in any sense be regarded as possessing any diagnostic importance.

VI. SUBPHRENIC PYOPNEUMOTHORAX.

In a noteworthy contribution recently published by Leyden attention is directed to subphrenic, air-containing **abscesses** of the abdominal cavity, for which he proposes the name of subphrenic pyopneumothorax. Owing to the similarity of the symptoms to those of pneumothorax, the condition merits the careful attention of diagnosticians. Although these air-containing abscesses were not unknown to earlier observers, the rational basis for their diagnosis was first created by Leyden.

These abscesses, as is well known, may be produced by any perforative processes in the region of the abdominal organs, whether in traumatic peritonitis or in other forms of peritonitis, perityphlitis, gastric or duodenal ulcers, perforating carcinoma, hepatic or splenic abscesses, rupture of the gall-bladder by gall-stones, or putrefying echinococci. Abscess in the subdiaphragmatic space may also represent a grave sequel of a small infectious pleuritic exudate.

Subphrenic abscesses may be divided by a median line of demarcation, formed by the suspensory ligament of the liver, into right-sided and left-sided abscesses, but this anatomic division, which cannot always be carried out, is of no practical value.

In the former class (right-sided abscesses) the lower border is formed by the upper surface of the downward displaced liver; in left-sided empyema the inferior and lateral boundary is formed by the stomach, colon, or spleen. If the enlargement of the cavity downward is hindered by peritoneal adhesions, and the pressure of the contained air is directed upward, the lower portions of the lungs present the characteristic appearances of pneumothorax, such as disappearance of the pectoral fremitus, metallic phenomena, especially on shaking the patient, and partial disappearance of the respiratory murmur.

The **diagnosis** will, of course, depend chiefly on the history of existing or antecedent disease of the abdomen, perityphlitis, disease of the liver, periphlebitis, etc. Other factors influencing the diagnosis are: relatively mild pain on pressure, absence of tension and bulging of the intercostal spaces, and a very slight degree of dislocation of all the organs. The absence of cough must also be considered, as well as the fact that normal vesicular breathing is heard over by far the greater part of the lung, this area being sharply divided below from the area of faint amphoric breathing, which, however, must be carefully distinguished from typical amphoric breathing.

Pfuhl suggests the use of manometric measurement as a diagnostic sign, the manometer being connected with the abscess by means of a cannula. The manometric pressure in the pleural cavity falls as the diaphragm is depressed during inspiration, and accordingly rises during expiration, while of course the opposite conditions prevail below the diaphragm; that is to say, the column falls during the expiratory rise of the diaphragm and rises during inspiration.

Unfortunately this sign is not quite trustworthy, because similar conditions may be observed in large pleural exudates which bring about a kind of tonic palsy of the diaphragm. Owing to the pressure of the intercostal muscles, the excessive action of which is not expended on the dilatation of the thorax, a slight inspiratory rise and expiratory fall of the manometer is noted which is in direct contrast to ordinary conditions.

VII. DIAGNOSIS.

The diagnosis of pneumothorax in the early stages and when the amount of air is small is not always easy, because the metallic phenomena, which constitute the most important signs, and the almost equally valuable results of percussion are apt to be ambiguous even when they are distinctly present, which is not always the case at first.

I have seen a whole series of cases in which there was collapse, dyspnea, disappearance of the respiratory murmur on both sides or only on one side, just as in pneumothorax, without the classic metallic phenomena making their appearance; and, as subsequent observation showed, the symptoms were not due to the escape of air into the pleural cavity but to obstruction of a pulmonary artery, acute emphysema, rheumatism of the chest muscles, cardiac weakness, etc. On the other hand, I have also seen cases suddenly develop metallic sounds with characteristic dyspneic phenomena, though subsequent observation showed that, instead of the serious condition of pneumothorax, one had to deal with acute dilatation of the stomach, or the sudden production of the metallic phenomena in a large cavity after the expectoration of large masses of sputum, or the rupture of a septum traversing the cavity.

To this ambiguity of the symptoms the further difficulty is in many cases added that, out of regard for the sufferings of the patient, the diagnostic methods cannot be utilized to their full extent because any change of position or bodily disturbance must be avoided for fear of enlarging an already existing perforation and increasing the weakness and collapse or aggravating the pain and dyspnea. A painstaking examination is therefore always to be recommended, and with some perseverance on the part of the examiner his end can usually be attained without doing the patient any serious damage.

It is not, as a rule, very important to determine whether the fistula of a pneumothorax is open or closed. It is much more important to *determine the primary disease*, so as to learn the cause for the escape of air into the pleural cavity. It should also be determined whether an exudate is present and whether the pneumothorax is completely encapsulated, as in the latter case I believe interference is positively contraindicated.

Notwithstanding the fact that the metallic phenomena are especially well marked in an encapsulated pneumothorax, this condition is very apt to be mistaken for a large cavity, especially as the position of the neighboring organs is, of course, not materially affected. While from a scientific point of view the sudden breaking down of a septum in the cavity, with the production of metallic phenomena like those seen in pneumothorax, may be of the greatest interest, such an event does not in the slightest affect the treatment of the case, for the diagnosis is of no use to the patient, since it is too late for any active interference.

The differential diagnosis between a large **cavity** and pneumothorax is purely a diagnostic feat, if we merely determine whether the metallic phenomena occurring in the course of advanced phthisis depend for their production on the former or the latter condition. As a rule, the nutrition of a patient presenting large cavities with distinct metallic phenomena is already seriously impaired, and if the general condition is fairly good, the existence of a large cavity can scarcely be taken into serious consideration.

On the other hand, it is very important to determine whether *tuberculosis* is the cause of a pneumothorax, for even a small tuberculous focus may rupture, and in such a case recovery both from the pulmonary disease and from the pneumothorax is not by any means impossible. Marked nutritive disturbances, the history of a former pleurisy, apical catarrh, swelling of the glands, hereditary predisposition, great constancy of the phenomena, positive result of an analysis of the sputum, which often contains numerous bacilli, and hemorrhages from the lung would, of course, tend to indicate the phthisical nature of the process.

It is of the greatest importance to determine whether pneumothorax is caused by a *gangrenous focus* in the lung or by a *putrid exudate,* for in the latter case operative interference is distinctly indicated, just as it is in that form of pneumothorax which is produced by the *rupture of an empyema* into the bronchi. But the treatment, to be successful, presupposes the greatest accuracy in diagnosis, hence all the phenomena must be tested with the most painstaking care. Examination of the sputum and exploratory puncture are the two methods that most strongly recommend themselves under these conditions, because the demonstration of tubercle bacilli, on the one hand, and, on the other hand, the finding of crystals of hematoidin and of fatty acids or of the characteristic offensive plugs and shreds of tissue, or even the mere presence of numerous pyogenic micro-organisms, is of the greatest importance.

According to Wintrich, it is almost impossible to mistake pneumothorax for an advanced degree of **emphysema**, as the signs of pneumothorax are much more intense and the physical signs vary widely in the two conditions. Nevertheless I have seen cases in which the diagnosis was at first anything but easy. The difficulty would not, of course, arise in simple emphysema with good compensation; it might, however, arise when there is a sudden acute pulmonary insufficiency, such as occasionally develops in the later stages of emphysema, or when a sudden intense enlargement of the lung is accompanied by extreme dyspnea. In such cases there is complete collapse, the cardiac dulness, owing to the extreme inflation of the lung, almost or completely disappears, the respiratory murmur is inaudible or replaced by a peculiar, faint amphoric breathing, especially in the interscapular space, and the sound obtained by percussion shows the highest grade of resonance. The diagnosis can, perhaps, be positively determined only by the symmetric appearance of the phenomena, since double pneumothorax is one of the very rarest of occurrences, and by the abnormally loud percutory sound over the entire thorax, combined with comparatively faint metallic phenomena.

Other factors to be considered in the diagnosis are a negative result with Heubner and Leichtenstern's method of percussion with the handle of the pleximeter, and the presence either of large mucous râles characteristic of bronchial catarrh, or of diffuse crepitant râles, indicating the beginning of pulmonary edema, which usually accompanies the pulmonary insufficiency.

Diaphragmatic hernia is a condition that is apt to be mistaken for pneumothorax; the most important signs in favor of hernia are violent abdominal pain, colic, vomiting, indicanuria, and albuminuria. The percussion note in hernia almost always, unless strangulation and extreme meteorism are present, has a distinct tympanitic character. In some cases the absence of fever will suggest the diagnosis, although subnormal temperature with symptoms of collapse and shock are not unknown in pneumothorax.

Another sign, contributed by Gerhardt, is that the râles in hernia are associated more closely with peristalsis than with the respiratory movements and are more continuous.

In addition to what has been said about the diagnosis of open and closed pneumothorax and of pulmonary fistula (see Mechanism of Escape of Air and Symptomatology), we may mention the following points:

Increased intensity of amphoric breathing does not always indicate the presence of a fistula; the amphoric breathing may become intensified whenever the media are unusually favorable for sound-conduction and the respiratory movement in the lung suffices for the production of a distinct respiratory murmur.

Nor does the periodic occurrence of *violent paroxysmal cough,* accompanied by the *expectoration* of large masses of exudate (Wintrich), appear to possess any diagnostic value for the presence of an open fistula. As has already been stated in the description of pleurisy, this copious paroxysmal expectoration does not always come from the pleura, as it may emanate from bronchiectatic diverticula which are evacuated either by the pressure of their own contents or, as occurs more frequently, by a change of position. This copious expectoration is not even a positive sign of the existence of a true bronchiectatic cavity, as the bronchial mucous membrane in the resting lung may produce enormous quantities of pus and mucus, which, owing to the diminished cough reflex, are only expectorated from time to time. The view advanced by various authors that the masses contained in the pleura are not expectorated until the level of the fluid, by a change of position, is made to exceed that of the fistula, appears to me to be in direct contradiction to the mechanism of the respiratory organs. Besides, the point where the air-current which constantly passes through the fistula exerts the greatest amount of irritation is where we are most apt to have the production of a solid exudate. The attempt has also been made to obtain some data for the diagnosis from the chemical composition of the air. Davy, Martin, Solon, Wintrich and Le Conte have devoted some study to this question, without, however, arriving at any definite conclusions. Quite recently Ewald again bestowed his attention on this question, and by means of a comparatively simple procedure obtained results which he believed justified him in stating that whenever the proportion of CO_2 in the air of a pneumothorax falls below 5% an open fistula may be positively assumed, while a proportion exceeding 10% indicates a complete occlusion (encapsulation), and a quantity varying between 5% and 10% an incomplete occlusion.

These figures do not appear to be sufficiently correct to afford a basis for a practical diagnosis, but a consideration of the general conditions and the local symptoms usually indicates whether the gas is still accumulating or has ceased to escape, although this does not, of course, mean that the fistula has become permanently closed.

VIII. DURATION, COURSE, AND PROGNOSIS.

The course of the disease is usually dependent on the primary trouble. In debilitated individuals, in advanced tuberculosis, when the neighboring organs are much displaced, the exudate accumulates rapidly, and there is diffuse or far-reaching gangrene of the lung, the result is almost always fatal. Death in such cases may ensue a few hours after perforation. It is certain to occur within a few weeks, with the rapid formation of a large purulent exudate, which quickly undergoes putrefaction. The fatal outcome is frequently preceded by rapidly developing edema in the lower extremities, congestion of the liver, ascites, and decubitus, and the patients suffer greatly from attacks of dyspnea and protracted paroxysms of cough.

According to Weil's analysis, one-fourth of all the cases of pneumothorax die within the first weeks, and one-half during the first month; by far the majority die a few months after the escape of air. Cases have, however, been reported in which death was delayed for many months or even for several years. [Morse concludes from his "Analysis of 51 Cases of Pneumothorax"* that recovery from the pneumothorax may occur in about 15% of all cases. The cases which recover are practically all cases of serous effusion, and the patients usually die later of pulmonary tuberculosis. The pneumothorax is the direct cause of death in 60% of all cases; 80% die in less than a year, and only 10% live over five years. The prognosis is worse in right-sided than in left-sided, and in purulent than in serous pneumothorax.—Ed.]

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* *Amer. Jour. Med. Sci.*, May, 1900.

Uncomplicated traumatic pneumothorax, on the other hand, as well as internal pneumothorax due to the mere rupture of an emphysematous area, has a relatively favorable prognosis. [Rose reports twelve cases of complete recovery from tubercular pneumothorax without effusion. There is distinct prospect of cure if the lungs are not greatly involved and no complication exists. The purulent cases are the most serious. Operation is advised whenever practicable, as it is always followed by improvement even when recovery does not occur.—Ed.] Pneumothorax following a solution of continuity of the chest-wall by gun-shot or stab wounds runs a favorable course unless there has been severe injury to the lung, or other complications arise. The smaller the amount of air escaping during the first two days, the more certain the prospect of spontaneous recovery. *Hemorrhage,* as a rule, does not affect recovery unfavorably unless it is profuse enough to weaken the patient or produce a lasting anemia. Indeed, a certain amount of hemorrhage appears to have a favorable influence in preserving asepsis within the cavity.

It goes without saying that the presence of **foreign bodies,** such as a knife-blade, projectile, pieces of clothing, or splinters of bone, materially affect the course of the disease and ultimate recovery, both by their mere presence and by the necessity to which they give rise of resorting to operative intervention. On the other hand, appropriate surgical interference may tend to arrest the irritation of the pleura and the formation of the exudate, so that the prognosis as just given applies only to spontaneous recovery.

In pneumothorax due to **perforating abscesses after pneumonia,** or to superficial **gangrenous foci** of embolic origin or secondary to pneumonia, the chances of recovery by operative means at least are comparatively favorable, provided, however, of course, that the infection becomes localized and not more than one purulent or gangrenous focus is present. The chances of recovery in such a case depend on the formation of dense adhesions about the infected focus and on the ease with which the focus can be reached for purposes of treatment (see page 950).

If, however, the empyema or pneumothorax collects at some point remote from the gangrenous focus, as sometimes occurs, the chances for recovery after operative intervention are less favorable; for this reason pneumothorax after extensive gangrene originating in more than one focus always offers a bad prognosis.

It is difficult to formulate any general statement in regard to the **prognosis** of pneumothorax as regards spontaneous recovery *in phthisical patients.* In them pneumothorax is always a much more serious condition than a serous or even a purulent pleural exudate; the latter not infrequently heals spontaneously, and may even temporarily arrest the cheesy and tubercular processes in the lungs, while pneumothorax always exerts an unfavorable influence on the disease, as in the great majority of cases it ultimately leads to putrefaction. Accordingly, I have rarely seen complete recovery from pneumothorax occur in phthisical patients; almost all the patients die of asphyxia or exhaustion; in the most favorable cases the symptoms progress slowly with a periodic increase in the accumulation of air and exudate.

This statement applies, of course, only to pronounced cases of phthisis; pneumothorax occurring in consequence of a small tubercular focus may be completely recovered from, and even the original pulmonary symptoms may eventually disappear entirely.

In *favorable cases* the exudate as a rule increases very slowly or not at all, but, owing to the peculiar alterations in the sound-conducting properties of the tissues and the ease with which metallic phenomena can be heard over a wide area, it is not always possible to determine the size of the exudate and the changes in volume that occur, even when relatively light percussion is used.

The presence of an exudate very rarely leads to *absorption of the pneumothorax,* although cases occur in which the exudate gradually grows until finally not a trace of air can be demonstrated. As in these cases the presence of air cannot be demonstrated even after the fluid has been evacuated, it seems right to assume that the air has been absorbed by the fluid or by the pleura itself.

In extensive pleuritic processes, whether they are dependent on tuberculosis or not, it is to be regarded as a favorable occurrence when both the exudate and pneumothorax remain stationary. The patients gradually learn to accommodate themselves to the new conditions, and may make comparatively large gains in strength and general nutrition. The outlook is particularly good when there is no fever; but persistent subnormal temperature is a very bad sign.

Quite frequently a change in the proportion between the exudate and the air is observed; the exudate may rise or fall and again become increased without any demonstrable cause.

No additional data need here be given in regard to the form of pneumothorax where a permanent external or internal fistula after a time develops, as in cases of delayed recovery after empyema operations, or after rupture of an exudate into the lung, as this accident and its causes have been sufficiently discussed in connection with the operation for empyema. A patient with such a fistula may under certain conditions live for years in comparative comfort; he finally dies with symptoms of cachexia, general anasarca, or as a consequence of amyloid and fatty degeneration of the organs.

IX. TREATMENT.

From what has been said a number of **indications** naturally present themselves. The most important is to support the patient's strength as much as possible in cases of collapse when there is violent pain and when the patient has constant attacks of cough and is in danger of being asphyxiated, and to diminish the abnormal reflex phenomena as much as possible. There is nothing better in such cases than a *hypodermic injection of morphin* of the proper strength, as this combines the effect of an antispasmodic and sedative in equal degrees. Quite often examination is impossible until the patient is brought under the influence of morphin.

Morphin may be confidently given even in cases of profound collapse, as it never fails to have a favorable influence, just as in cases of stenocardia. There is no objection to giving, in addition, an antispasmodic such as *ether, wine,* or *coffee,* providing the patient is able to swallow. It will always be found, however, that if there is any possibility at all of reviving the patient's strength, nothing will accomplish it better than a morphin injection of the proper dose; given by the mouth, morphin has not anything like the same effect. The proper use of morphin enables one to dispense with the application of antipyretic and antiphlogistic remedies, which are usually of no avail, or of ice-bags, warm compresses, or cups.

It has been recommended that the diseased site be *bandaged* either with roller bandages or adhesive strips in order to immobolize it and counteract the pressure of the air; but this method of treatment is no more useful in pneumothorax than in pleurisy, as the necessary amount of fixation and counterpressure cannot be achieved without injuriously interfering with the breathing of the other side and causing the patient a great deal of discomfort.

The treatment in **traumatic pneumothorax** cannot be laid down dogmatically. If there is a penetrating wound of the chest with a large accumulation of air, and it is positively determined that carriers of infection or large foreign bodies have entered the pleural cavity, it is always advisable to *enlarge the wound and irrigate* thoroughly with a disinfecting solution and then to treat the condition, like any empyema, with *iodoform.* The earlier these measures are carried out, the better the prospect of success. Once the infection has become general, so that micro-organisms become deposited in the tissues, there is little chance of permanently removing the noxious material by means of irrigation.

When the amount of air is inconsiderable and the entrance of foreign bodies can be excluded by the character of the wound and the condition of the clothing, and, finally, in all cases in which there is no visible external wound, it is better to desist from any form of operation, or at most to make a *simple incision,* if, in spite of hypodermic administration of morphin, alarming symptoms of asphyxia from pressure on important organs make their appearance. At all events, it is advisable to *aspirate* the air once or twice just sufficiently to bring about a slight diminution of the alarming symptoms.

If a **hemothorax** exists at the same time, operation, as has already been stated, is indicated only when it becomes evident by the increasing pallor of the patient and by the other symptoms that the hemorrhage is still going on and is presumably arterial, as, for instance, from one of the intercostals which may be situated near the external wound and may be more accessible from within than from without. [G. N. Pitt reports a case of hemopneumothorax apparently due to the rupture of an emphysematous bulla.* The right pleural cavity, at the autopsy, was found to contain eight pints of fluid blood and clots, with much air under tension; the lungs were compressed but healthy, except for a small emphysematous bulla with an imperfect wall, near the right apex.—Ed.]

If it is positively known that the pneumothorax is due to **rupture** of an **empyema** or of a **gangrenous focus,** it is advisable, especially in the latter case, to *operate* at once. Unless there are dangerous complications the operation will almost always be successful. If it can be positively determined that an empyema has ruptured through the bronchi, an empyema operation is to be advised, in spite of the fact that spontaneous cure in these cases not rarely occurs; because operation, provided the lung retains some power of expanding, is certain to hasten recovery and is often the only means of curing the patient. To depend on spontaneous cure in such cases is, in our opinion, not rational, as the usual results of pleurotomy present better chances for recovery than the uncertainty of internal evacuation which fails to guarantee thorough elimination of all the pus and permanent asepsis. On the other hand, after rupture of a pulmonary ulcer into the pleural cavity the operation may be delayed, as in such cases, in spite of the entrance of air, there may be no decomposition and the air in the pleural cavity may undergo spontaneous absorption, especially if there are adhesions. The operation must always be performed if the symptoms are such as to indicate that the contents of the stomach or the intestines have entered the pleura, or if a rapid increase of the exudate and other alarming symptoms occur.

If it appears that the pneumothorax has been produced by a **foreign body entering from the esophagus or trachea,** operation is absolutely unavoidable, as the mere presence of a sharp object, such as a bone splinter or pin, is a constant menace, even granting that complete asepsis can be maintained, which is exceedingly improbable.

In the forms of pneumothorax which suddenly appear without warning and in an apparently healthy lung or after great exertion, there can be no thought of operation unless the patient's life is threatened by the formation of a large purulent exudate or the course of the fever is such as to suggest putrefaction. In such cases an exploratory puncture is, of course, indispensable.

With Leyden, I do not regard a **simple seropneumothorax** as an indication for incision, as serous exudation appears to be an expression of a peculiar form of irritation of the pleura which cannot be successfully combated by mere evacuation of the exudate. In **empyema,** on the other hand, the exudate itself, in the great majority of cases, is the most important irritant, and alone tends to keep up the inflammation in the pleura; hence the *empyema operation,* which has for its object the complete removal of this irritant, is usually followed by a very favorable result. As long as the exudate of a pneumothorax remains serous, therefore, there is no urgent need of operation, especially if it is accompanied by high fever, the chief sign of pleural irritation. In any case, however, the condition of the pulmonary tissue will eventually decide as to the necessity of operation.

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* *Clinical Soc. Trans.*, vol. XXXIII.

The question of operation is difficult to decide in all cases **complicated** by advanced destructive bronchopneumonia or emphysema, not to mention tuberculosis, for it is hard to see what good it can do to evacuate the air through the incision in diffuse degeneration of the pulmonary tissue.

That no radical operation should be undertaken in cases of advanced phthisis goes without saying, as the chances for the alleviation of the symptoms or even recovery bear no proportion to the direct consequences and hardships of an incision.

The practice of performing an incision to save life is in general to be condemned, as it is impossible to be sure beforehand that the lung, under the influence of the artificial external pneumothorax, will not retract and lose its function altogether.

Incision is therefore recommended under the following circumstances, providing the state of the lungs and the general condition of the patient are not absolutely unfavorable: when the exudate is very large, when the pus, as shown by the exploratory puncture, is thin, and when there is the slightest tendency to putrefaction. If these indications are observed, the radical operation will be followed by relatively favorable results.

Whether there is a **valvular pneumothorax** or a **closed pneumothorax** in the organic or mechanical sense, operation is equally out of the question, as in our opinion, the presence of an external wound can only have an unfavorable effect on the respiration without offering better chances for the permanent closure of the fistula, since the air-pressure of the pneumothorax itself effects the greatest possible increase in pressure obtainable, and must, therefore, eventually bring about mechanical closure of the fistula.

Puncture should be resorted to only in desperate cases; it will always be found that it is even less successful in pneumothorax than in empyema. Although the immediate relief may be quite considerable, it is never lasting; and if the exudate is at all large, two or three repetitions of the procedure, even when carried out with the greatest care, are practically certain to produce putrefaction and lead to rapid loss of strength. The operation ultimately loses even its momentary good effect, and I entirely agree with Senator, who believes that the palliative value of puncture in most cases is quite insignificant. It should be resorted to only when there is a vital indication; in all ordinary cases it is best to administer narcotics, which do a great deal toward removing the most distressing symptoms. If it can be positively determined that one has to deal only with an encapsulated or circumscribed pneumothorax of moderate extent, there is no objection to aspiration, but this diagnosis is very much harder to make in practice than on paper.

The **technique** of the operation is very simple; it can be done either by immersion in antiseptic fluid and with a simple fountain syringe, or with weak aspiration. With the first method it is, of course, quite common to have a smaller amount of air escape than when aspiration is used, and sometimes the behavior of the air may serve to indicate the degree of pulmonary expansion or the existence of paresis of the chest-walls and a relaxation of the tonus in the structures surrounding the pleura.

The puncture should be made in one of the upper intercostal spaces in the anterior or lateral wall, or the needle may be introduced at the point where the percussion note and metallic sound are loudest. I have never seen subcutaneous emphysema follow the operation unless it already existed in some degree from necrosis of the costal pleura. Unless the needle is left in place too long, or the edema and infiltration of the chest-walls are such as to produce a sinus with rigid walls that refuse to collapse, there is no danger of subcutaneous emphysema.

After many years of observation I have not been able to convince myself of the value of puncture and the injection of antiseptic or aseptic solutions in uncomplicated purulent exudates, and except in very rare cases, there is no reason to expect any better success from the same measures in pneumothorax. This view has been fully borne out by my later experience, and other observers also have come to the same conclusion.

The view advanced by Potain, that the chief danger in pneumothorax depends on the formation of large fluid and putrid exudates, is probably correct in the main, since, as has already been mentioned, the mere entrance of air from the lungs is a very small factor if the pleura is otherwise free from irritation and no other complications exist. His further conclusion, however, that the chief danger in pneumothorax can be removed by substituting sterilized air for the exudate is not admissible. In many cases, and especially when the exudate is not putrid, the danger does not lie in the exudate itself, but in the fact that the pleura happens to be in a state of irritation favorable for the production of a very large exudate; and although a return to normal conditions may be hastened and resorption possibly facilitated to a certain extent by at once replacing with air the fluid removed by the aspirator, I do not believe that the procedure is sufficiently radical to take the place of incision. Aside from the fact that the exudate cannot be completely evacuated, and that mere evacuation has no effect on the micro-organisms residing in the tissues themselves, it does not seem likely that if the pleural tissue is incapable of absorbing the watery constituents of the exudate, it should be capable of absorbing air, at least not to the extent necessary to bring about a complete cure. For these reasons the procedure advocated by Potain on the basis of an insufficient number of clinical observations does not appear to represent a very promising mode of interference.

The nutrition of the patient and his general condition must receive careful attention in all those cases which bid fair to run a chronic course, and a certain amount of *respiratory gymnastics* should also be prescribed in suitable cases. But if the local changes have already produced an intense disturbance of the respiratory mechanism that cannot be corrected either by operative intervention or by internal medication, such measures will not contribute much to the patient's recovery.

Whether *systematic feeding* of the patient based on scientific principles, to which Leyden attributes so much importance, has any favorable influence on the tuberculous process seems questionable. There is no doubt that proper feeding is most important for strengthening the constitution, but in my experience the elementary principles of dietetics have not as yet been scientifically formulated. I say this in the face of the great interest with which metabolic changes are nowadays being investigated, and in spite of the accuracy of the dietetic rules to which they have given rise. I find that we still have to fall back on certain stereotyped or purely empiric methods, and that in general the patient gets along best if his wishes are consulted to a certain extent without any theoretic prejudices, or at least if the physician by careful examination tries to find the true reason and correct interpretation of the patient's wants and wishes, which he quite frequently does not clearly understand himself.

Every physician of experience and every practical nurse will admit that more can be accomplished by coaxing the patient, by frequently changing his food, and by giving him the right thing at the right time, than by prescribing a rigid diet list. We have learned by experience that it is not always necessary to condemn a patient, when he has fever, to a starvation diet; but it would be just as wrong to allow a full diet to all fever patients because some of them happen to have a good appetite as it would be to starve every patient because many of them have no appetite and are unable to assimilate food. While it may, of course, be injudicious to place all debilitated patients on an exclusive albuminous diet, consisting of roast meats, broths, and eggs, or to allow them strong wines, it is equally wrong to deny such articles of food to a feverish patient if he happens to have an appetite for them, simply because it is generally taught that only cabohydrates and non-stimulating foods are appropriate for patients suffering from fever.

I do not believe, therefore, that systematic feeding consists in forcing a certain diet on a patient or in insisting on fattening him against his will, for I have often convinced myself when attempting to carry out forced feeding (*Mastcur*) or the so-called *gavage* that excessive feeding is not identical with sufficient nutrition; that is to say, the power of obtaining sufficient energy for maintaining existence and performing work. Fattening is not necessarily the same as strengthening the organism. I do not deny that a very nervous or melancholy patient must be fed, against his will if necessary, for the body requires the food even though the initiative for ingestion is wanting. But a physically healthy patient, as a rule, may be trusted to make known his wants. Even when a patient is unconscious, he will swallow liquid food if it is given to him in the proper way. I must, therefore, take exception to any form of forced feeding, by whatever name the method may be known, although I am quite free to admit that such a treatment may bring about a general improvement and increase in body-weight in certain forms of nervousness, in anemia, and in hysteria. This improvement is, however, in great part due to the absolute rest in bed and other changes in the manner of living which necessarily accompany the forced feeding, at least I have often been able to bring about a very satisfactory improvement in such persons simply by ordering them to bed for two weeks.

It is also to be remembered that in almost all cases of forced feeding the bowel movements are excessively copious, and largely consist of undigested masses of food, showing that excessive feeding is accompanied by a good deal of unnecessary work on the part of the digestive tract, and that the method, therefore, is adapted only for persons with sound organs in the medical sense, but with an abnormally reacting nervous system. A diet that may be appropriate for nervous and anemic individuals is not necessarily adapted to the wants of patients suffering from fever or organic disease.

It is true, as Leyden has pointed out with perfect justice, that in the case of phthisical patients, and, it might be added, other patients as well, an increase in body-weight indicates an improvement in the general condition, and, conversely, a loss in weight is a sign that the general condition is suffering; but one is not justified in concluding from this fact that the patients are better because their body-weight is increased; on the contrary, the natural conclusion is that the body-weight shows a noticeable increase because the plastic power of the organism, which is the sign of an increase in essential energy, has been restored or even augmented. The form of energy which we are now considering is not, in my opinion, obtained directly from the food; for if that were true, it would be impossible for the greatest athletic feats to be performed with a relatively low amount of food ingestion. An excessive ingestion of food merely tends to the production of fat and plays a very inconsiderable part in developing unusual powers of assimilation and muscular power in the body.*

What has just been said is not intended to belittle the value of rational dietetics; it is only meant to show the great paucity of our knowledge on the subject of energy production and the difficulty of deducing any definite rules of feeding.

The maintenance of the work of the organism is not determined merely by the amount of energy-producing material, so that the proper balance cannot be deduced from the amount of nitrogen, carbohydrates, or oxygen ingested and excreted. It is determined by the possibility of a profit, the power of the body to accumulate energy in excess of its needs; or, in other words, of keeping on hand an extra supply of all forms of vital energy by the conversion of a greater amount of energy-producing material than that which is lost in using up vital energy and preserving the cohesion and motion of the parts (attraction and repulsion, latent energy and reserve force); this, being accumulated as in a storage battery, becomes available at the required time for the production of work.

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* O. Rosenbach, "Die Entstehung und die hygienische Behandlung der Bleichsucht," Leipzig, 1893.

Thus two individuals in a state of nitrogen equilibrium, while apparently possessing the same balance, do not by any means show the same profit; one of them, for instance, may, after the work has been performed, exhibit a greater relaxation of tonus or power of adhesion between the different parts of his organism. In such a case the same amount of work is performed at the cost of using up the machine (impairing its power of cohesion), and this factor, which in a commercial balance is taken account of under the name of wear and tear, deserves more careful consideration than it has so far been accorded in investigations of metabolism which simply represent the determination of the proper balance—the relation between receipts and expenditures, wear and tear and profit.

The advantage gained by feeding a patient suffering from pneumothorax and excessive dyspnea with an esophageal tube, which avoids the necessity of active muscular movements, appears doubtful, since a large part of the muscular movements can be equally well avoided by simply pouring liquid food into his esophagus. Dyspnea is excited and aggravated not only by the movements of the trunk and extremities associated with the taking of food, but also by the reflex effect of the act of deglutition on respiration and circulation, and there does not seem to be any doubt that gavage and forced feeding in themselves, by overloading the stomach, directly increase the mechanical demands on the respiration, not to mention the fact that the chemical elaboration of the food-stuffs which requires an extra supply of oxygen also demands an increase in the respiratory rate.

Both on theoretic grounds and after a large personal experience I therefore conclude that in pneumothorax, whether dependent on tuberculosis or on chronic pneumonia, no advantage is to be derived from any form of *Mastcur*,—to use this term which best expresses the true object aimed at, namely, excessive feeding,—although I regard rational feeding as one of the most important therapeutic principles, which deserves to be studied from an entirely new point of view.

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